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THE AIR WE BREATHE.[1]

By ALAN BELL, M.B., B.S., D.I.H.,
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. . . For over this vast city, in size, wealth and power the greatest communal achievement of a man's sojourn on the planet, had fallen a perpetual pall. The classical pillars and ornaments of the churches and large buildings were half hidden under soot: the naked Achilles in the park, tribute to the Iron Duke, was almost black. Even the dripping trees and foliage were grimy. It was like Homer's Hall—the land of the Cimmerians. . . . The vast space, which is the South, stretched between the earth and sky cannot be described . . . there is no air; there is nothing but liquid fog. . . .[2]

There are numerous indications that the general public of New South Wales is becoming increasingly aware of the importance of clean air. However, much remains to be done; health inspectors require extensive and up-to-date knowledge of this subject if they are to play a major role in stimulating local interest and action. In the short time at my disposal I should like to describe local conditions and discuss the apparent importance of low concentrations of sulphur dioxide.

Severity of Atmospheric Pollution.

In 1953 the Division of Industrial Hygiene, Department of Public Health of New South Wales, started a deposit gauge survey, and within a short time 63 stations were established in Sydney, Port Kembla and Newcastle. Monthly dust fall measurements are made in a manner similar to that used overseas. Gradually the scope of our work is being enlarged as new equipment is received, and it is hoped that within a few years we shall be able to fill in many of the present-day gaps in our knowledge.

Topographically the three main manufacturing centres of New South Wales are advantageously situated in respect of atmospheric pollution; they are near the coast, and in some months prevailing winds considerably reduce the severity of this social evil. Moreover, because of favourable climatic conditions only 2% of the total amount of coal mined in New South Wales is used for domestic purposes. The majority of this is used in mining towns which are not large, nor are they major manufacturing centres. The degree of dust fall in 1954 is shown in Table I.

Although New South Wales is a young State from the point of view of industrialization, it is unfortunately true that in some respects the major cities of this State have

[1] An address given to health inspectors at the Health Congress of the Royal Society for the Promotion of Health, on November 23, 1956, at Medlow Bath, New South Wales.

[2] This description of mid-nineteenth century London is given in "English Saga 1840-1940", by Arthur Bryant.

problems comparable with those found in many large overseas manufacturing centres.

The Role of Sulphur Dioxide.

After reading the literature on atmospheric pollution, one is aware of the incompleteness of existing knowledge. Major serious difficulties are encountered whenever investigators try to determine whether or not "day to day" air contamination has an adverse effect on the health of the general population. However, by studying reports of the recent overseas "smog" catastrophes it is possible to paint a few broad strokes on the canvas. As knowledge increases it may be necessary to modify the general outline of the main picture; at this stage a considerable number of leading world authorities consider that in many countries small concentrations of sulphur dioxide are of considerable importance.

London smogs have a world-wide reputation. The last two tragedies occurred in December, 1952, and January, 1956. In 1954 the Ministry of Health isssued a report which is full of interesting data. The public was rightly alarmed when it realized that the 1952 fog caused "between 3500 and 4000 deaths", details of which can be seen in Table II.

TABLE I.

Location.	Type of District.	Total Insoluble Material (Dust, Soot, Ash) Expressed in Tons per Square Mile per Month.
Sydney	City area	25·6
	Industrial	17·7
	Inner residential	8·0 to 10·0
	Outer residential	5·0 to 3·0
	Pyrmont (an area around the dock and coal fed electricity generating station)	46·1
Newcastle ..	City area	25·9
	Industrial	12·4 to 35·8
	Outer residential	6·5
Port Kembla ..	Industrial—near steel works ..	36·3 to 57·9 (individual readings occasionally reach 140·0)
	Residential	17·5 to 23·2

Under the heading "Delayed Effects of Fog", the authors state that many people "though apparently recovering from the acute illness of the December episode, may have been left with some degree of damage to their respiratory or cardiac systems causing them to succumb the more readily during a subsequent illness. . . . An analysis of the causes of death shows that of the increase of 1,539 deaths during the week ended 13th December, 1952, over those of the preceding week, 1,248 or 81 per cent. were due to diseases of the respiratory and cardiovascular systems. . . . Respiratory diseases (mainly bronchitis) alone during the same week accounted for nearly 59 per cent. of the total increase and during the week ended 20th December for more than 76 per cent. . . .". Some of those who died had not been outside their homes during the period under discussion; this was also the case in Liège in 1930, when a similar pollution disaster occurred (Haldane, 1931). A statistical analysis of the London deaths revealed that the very young and the older age groups were those most seriously affected. One should not forget that the increasing use of chemotherapeutic and antibiotic agents has had the effect of extending the life of many people suffering from chronic geriatric diseases.

During and immediately after the fog, arrangements were made for an additional number of autopsies to be performed. The findings (Ministry of Health Report, 1954) "were indistinguishable from those found after death from similar causes before the fog . . . there was no lesion or grouping of lesions which could be said to be pathognomonic of death due to fog". When the deaths were sorted into social classes (Leff, 1953) it was not possible to find any deviation from the normal pattern.

The Ministry's report shows how the evil effects of the smog were seen in many other ways; for example, the sickness claims in London and Middlesex were 9000 in excess of the expected number, and "acute" admissions to hospitals rose from 750 to 1100 a day. During the period under discussion the demand for the services of general practitioners considerably increased; one of them (Fry, 1953) wrote an excellent clinical description of the condition of his patients.

In 1953 the Committee on Air Pollution (chairman, Sir H. Beaver) presented an interim report to Parliament. In this document the following statement appears:

> We are not in a position, nor is there the evidence, to enable us to identify with certainty the pollutants and the combination thereof which were responsible for the particularly harmful nature of the London smog. There is, however, a clear correlation between the pollution by smoke and sulphur dioxide and the daily death rate in Greater London at the time. . . . Both medical opinions and chemical investigations indicate that the harmful effects of oxides of sulphur are greatly enhanced by the presence of smoke particles.

The committee's final report (1954) is in a similar vein.

The following chart (Figure I), produced by the Fuel Research Station, and taken from the Interim Report of the Committee on Air Pollution (1953), shows the similarity in pattern between the number of deaths and the rise in sulphur dioxide concentrations.

The highest recorded concentrations of sulphur dioxide were 1·339 parts per million on December 7 and 8. The corresponding figure for smoke was 4·46 milligrammes per cubic metre. In all probability substantially higher values existed for short periods of a few hours. Normal winter "concentrations of SO_2 in the air of towns near ground level may be 0·17 to 0·35 p.p.m. by volume of air" (Committee on Air Pollution, 1953).

It is known that the death rate from bronchitis per 100,000 is approximately 50 times higher in England and Wales than in comparatively little industrialized countries such as Denmark and Norway (Committee on Air Pollution, 1954). Pemberton and Goldberg, of the Department of Social and Industrial Medicine, University of Sheffield (1954), state that, after showing a steady and marked increase in death rate from bronchitis in passing from rural areas to the towns, cities and conurbations, "the evidence supports the hypothesis that sulphur dioxide air

TABLE II.

Number of Deaths During the Period November 15, 1952, to January 10, 1953.

London Fog of December 5 to 8, 1952.	Deaths Registered During the Week Ended								
	November 15	November 22	November 29	December 6	December 13	December 20	December 27	January 3	January 10
Greater London (population 8,364,000)	1565	1699	1902	2062	4703	3138	2234	2977	2634
(Average corresponding week, 1947-1951) ..	(1747)	(1708)	(1809)	(1805)	(1852)	(1914)	(1923)	(2303)	(2213)
London Administrative County (population 3,363,000) ..	747	753	853	945	2484	1523	1029	1372	1216

pollution is a factor in determining the magnitude of the bronchitis death rate of middle-aged and elderly males". They admit that other factors, such as occupation, may be concerned. D. D. Reid (1956) recently commented that "the rate of invaliding from the postal service in London because of bronchitis is considerably higher among workers in the central area and North East quadrant than for men in other London districts; this fits in with the fact that the prevailing south-westerly wind causes a build-up of air pollution in these areas".

The evidence incriminating sulphur dioxide has led to the current experimental use of "anti-smog" bottles. These bottles contain a solution of ammonium; when they are required the wick is pulled up and this releases fumes which in turn neutralize the acid in the air. This development arose from the observation that at the 1952 Smithfield Show prize catttle died from the smog, whereas neither pigs nor sheep were affected. The latter were

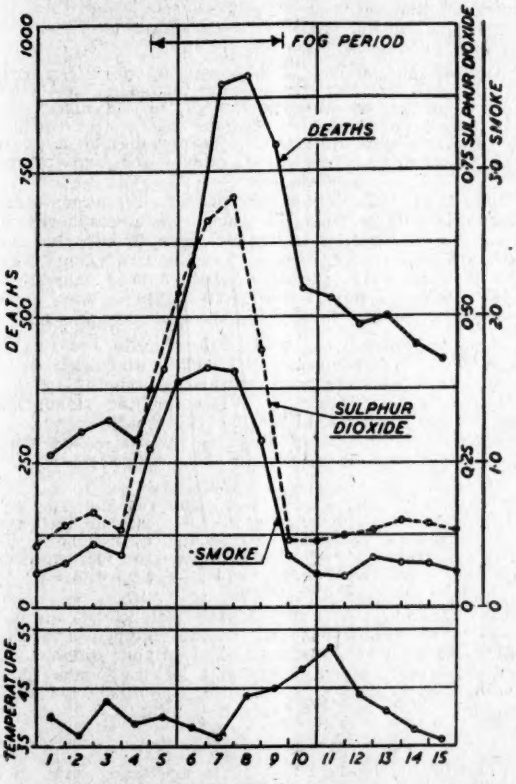

FIGURE I.

Deaths, air pollution and temperature during December, 1952, in Greater London (population 8,500,000). Sulphur dioxide: concentration in parts per million of air (mean of 10 sites). (Note: one part per million parts by volume of air equals 2·86 milligrammes of sulphur dioxide per cubic metre of air at normal temperature and pressure.) Smoke: concentration in milligrammes per cubic metre of air (mean of 12 sites). Temperature: degrees Fahrenheit (noon readings on Air Ministry roof). Deaths: total number occurring each day. (From Committee on Air Pollution Interim Report, 1953.)

housed in contaminated quarters, and it is thought that the ammonium given off from their excreta protected them ("The Anti-Smog Bottle", 1955).

It has been known for many centuries that sulphur dioxide is an upper respiratory tract irritant. Ramazzini, in his famous disquisition *"De Morbis Artificum"* written in 1713, recounts how a baker "barely escaped falling dead on the spot" when trying to extinguish "sulphur balls used for lighting fires in his oven". The victim was "tormented for many days by a distressing cough and had great difficulty of breathing".

According to Henderson and Haggard (1943), the physiological responses to various concentrations of sulphur dioxide are as follows:

	Parts of sulphur dioxide per million parts of air.
Least amount causing immediate irritation to the throat	6 to 12
Least amount causing immediate irritation to the eyes	20
Least amount causing coughing	20

For industrial conditions the maximum allowable concentration for this gas for an eight-hour working day is 10 parts per million ("Threshold Limits for 1956"). This standard is not a suitable one to apply to a heterogeneous population. Recent experimental work suggests that concentrations much lower than those previously mentioned may cause disease.

Because the human system can compensate to a remarkable degree, minor changes in the health of individuals are often difficult to measure; this adaptability is less evident at the extremes of life. Foulger (1951) made the following statement:

> It is a fundamental principle of general physiology that the imposition of a physiological stress upon any living organism leads to adjustment of the internal environment including changes in respiration rate, blood flow, blood pressure to counteract the stress. If the stress is of low intensity the organism may be able to meet it on a "pay as you go" basis, and no significant fundamental changes will occur. But, if the stress is most intense, the internal readjustment cannot take place with sufficient rapidity . . . The rate of recovery will depend upon the degree of deterioration and the recuperative power of the organism . . . Repetitions of the same degree and duration of stress before recovery is complete will catch the organism in a most vulnerable state and lead to an ultimate degree of deterioration greater than that produced by the initial imposition of stress.

It is unfortunate that the responses to low concentrations of sulphur dioxide are frequently not easily detected except under experimental conditions.

In Amdur, Melvin and Drinker's experiments (1953), 14 healthy resting men breathed, for short periods, varying concentrations of sulphur dioxide, from one to eight parts per million, through a face mask. The pulse rate rose by eight or nine beats a minute, the respiratory rate increased by three to four breaths per similar period of time, and the tidal volume fell to a level approximately 25% below the control value. Similar results were not obtained when factory employees were tested; during the course of their work these men were frequently exposed to 10 parts per million or more. Accordingly, it would appear that the reaction of workmen accustomed to an irritant gas is not the appropriate standard by which to judge the reaction of an unacclimatized subject. Lawther (1955) criticized certain aspects of this work and was unable to obtain similar results; in his experiments 10 parts per million produced a considerable bronchospasm in one subject.

Amdur and Mead (1955) also experimented on unanæsthetized guinea-pigs which, when exposed to sulphur dioxide (range 15 to 44 parts per million), showed a decrease in compliance, alteration in tidal volume change and an increase in resistance change. Amdur makes the following statement:

> From the point of view of both physiology and toxicology the resistance change is the most important of the criteria examined. It may be taken as an index of bronchial constriction. . . . The increase in resistance to air flow and secondary changes impose an added load of respiratory work on the animal even though the external symptoms are not discernible.

It is obvious that an increase in breathing which is of no consequence to a normal subject may well overtax a debilitated person. This research worker also makes the following statement:

> We have demonstrated that SO_2 produces bronchial constrictions in an animal and that these changes can be related to concentrations.

Sulphur dioxide, therefore, cannot be dismissed as only an irritant of the upper part of the respiratory tract.

> The effect of 25 p.p.m. reaching the lungs directly is of the same order of magnitude as that of 100 p.p.m. entering the upper air way.

This is a very important point and will be further discussed in a few moments. One part per million of sulphur dioxide oxidizes to sulphur trioxide, which can combine with water to form an amount of sulphuric acid equivalent to four milligrammes per cubic metre. The hourly conversion rate is 0·1% to 0·2% in natural sunlight. In 1952, Amdur, Silverman and Drinker showed that the breathing of air containing sulphuric acid mist in concentrations of 0·35 to 0·5 milligramme per cubic metre (0·12 to 0·17 part per million) for periods of twelve minutes would also cause shallower and more rapid respiration in healthy people. At the lower concentrations the subjects were not aware of the presence of the acid by any sensory response.

Another article (Cralley, 1942) discusses the important rolè of the ciliated epithelium in the removal of inhaled foreign matter from the upper part of the respiratory tract. Pieces of excised trachea from recently killed rabbits were observed when exposed to varying percentages of sulphur dioxide. The results showed that the time taken for the activity of the cilia to cease depended upon the amounts used. The following conclusion was reached:

> The concentrations which produce immediate irritation of the throat offer a comparative index of the deleterious effect of these gases upon ciliary activity when the exposure is for a short period of time.

Modified, but similar, experiments were conducted on human volunteers: a red dye was placed on the active region of the inferior meatus of the nose, and the times for its removal were measured when the patient was exposed to varying concentrations of sulphur dioxide. It appears that "Exposure to small amounts of irritant gases for long periods of time may lead to a chronic irritation of the mucosa which would accordingly affect the ciliary activity". I consider that any agent which decreases the effectiveness of a defence mechanism of the body is important, as it may lead to major consequences in susceptible people.

The state of our knowledge is such that it is not clear or easy to explain how low concentrations of sulphur dioxide cause trouble. Professor N. Nelson (1955), in trying to offer an explanation, stated that "it may be anticipated that irritants acting together may yield at least an additive field". Workers at the Stanford Research Institute, United States of America (1949), have made similar comments. After listing the various components of Los Angeles smog, they point out that when each constituent was tested individually, "none of these substances used in concentrations actually found in air (less than 0·6 p.p.m.) caused typical eye irritation, but together these substances definitely cause irritation".

A similar view was expressed in a leading article of the well-known journal *Industrial Medicine and Surgery* (1950):

> Traditions die slowly in any land and in any profession . . . One which lingers on is the belief that any harmful substance in the atmosphere acts solely through its own properties and in conjunction with no other substance that may co-exist . . . In the case of atmospheric pollution due to smog . . . no one has been able to detect any level of any one substance conducive to the harm that manifestly has been brought about. At the same time, examining toxicologists may have reported the presence of eight or ten agents in trace quantities well below accepted dangerous levels. It then becomes pertinent to inquire if the possibility does not arise that two or more, acting synergistically, fully may account for the evil consequences. A somewhat dissimilar possibility presents itself in the concept that some one of several atmospheric constituents may serve as a "trigger" that imparts to one or more other elements a celerity or quality of action not known when solely present.

A synergistic effect occurs when guinea-pigs are simultaneously exposed to 89 parts per million of sulphur dioxide and eight milligrammes per cubic metre of sulphuric acid mist. The effects of the mixture on growth, lung disease and respiration are more pronounced than the effects of either used alone (Amdur, 1954).

Other research workers (Dautrebande and Capps, 1950) have shown that in some instances aerosols can also exert a synergistic effect. For example, if aerosols are present

> . . . in the form of 1 to 2·5 p.p.m. of sodium chloride or 1·5 to 4 p.p.m. of used motor oil or 0·07 to 0·3 p.p.m. of 10% suspension of carbon black, they greatly increase the irritant activity of 0·55 p.p.m. of SO_2 on the eyes, nose and throat. Since the sodium chloride was non-irritant and the motor oil and the carbon black in diesel oil had very little action, it appeared probable that a truly synergistic effect was involved.

Other experimenters (La Belle *et alii*, 1955) placed animals in a chamber into which an aerosol and vapour could be blown independently by separate tubing. The survival times of the animals were measured when they were exposed to varying types and concentrations of fumes and aerosols. The results were as follows:

> The effect of aerosols in altering the toxicity of irritant gases is closely and directly related to the relative penetration of particles and vapour molecules. When the aerosol penetration exceeds that of the vapour the toxicity is increased. . . . There must be a physical combination between the vapour and aerosol or no change will occur at all. . . .

Industrial medical literature provides numerous samples of additive effects in fields other than atmospheric pollution. For example, it has been shown in experiments on mice (Chernov and Liberman, 1947) that "neither CO alone nor benzene alone caused a very marked mortality at certain temperatures. But when the animals were exposed to the two together the cumulative toxic effect was notable".

A little while ago I made reference to the fact that "the effect of 25 p.p.m. of SO_2 reaching the lungs directly is of the same order of magnitude as that of 100 p.p.m. entering the upper air way" (Amdur and Mead, 1955). Some authorities postulate that this could happen as a result of the absorption of gases by particulate matter. Professor N. Nelson (1955) wrote as follows:

> Particulates, in the size range below 1 micron diameter, have a fair likelihood of reaching the depths of the lungs and being deposited there. In the case of a gas like SO_2, it can be estimated that, if borne in or on particles of the order of 0·2 micron diameter, up to five times more might reach the deeper tissues than if the material were inhaled as a gas.

Hemeon (1956), an internationally well known authority, belittles the importance of this mechanism and quotes figures to show that the amount of sulphur dioxide which can be transported to the depth of the lungs in this manner is extremely small and would not cause any reaction.

Conclusion.

In spite of the fact that there are many gaps in our knowledge, many countries have pushed ahead with legislation to control atmospheric pollution. If industrial hygienists had waited the required number of years until all the contributing factors of any given problem were clearly understood before instituting control measures, then industrial hygiene would not be so far advanced as it is today. There can be no doubt that the effects of atmospheric pollution on health are wholly bad, whether measured in terms of well-being, of joy of living, of disease, or even of death.

Acknowledgement.

I wish to thank Dr. H. G. Wallace, Director-General of Public Health, Department of Public Health, New South Wales, for permission to publish this paper.

References.

AMDUR, M. (1954), "Effects of a Combination of Sulphur Dioxide and Sulphuric Acid on Guinea Pigs", *Public Health Rep.*, 69: 503.

AMDUR, M., and MEAD, J. (1955), "A Method for Studying the Mechanical Properties of the Lungs of Unanæsthetised Animals: Application to the Study of Respiratory Irritants", Proceedings of the 3rd National Air Pollution Symposium, sponsored by Stanford Research Institute: 150.

AMDUR, M. O., MELVIN, W., and DRINKER, P. (1953), "Effects of Inhalation of Sulphur Dioxide by Man", *Lancet*, 2: 758.

AMDUR, M., SILVERMAN, L., and DRINKER, P. (1952), "Inhalation of Sulphuric Acid Mist by Human Subjects", *Arch. Indust. Hyg.*, 6: 305.

"Anti Smog Bottle" (1955), *Smokeless Air: The Smoke Abatement J.*, Winter: 106.

CHERNOV, V., and LIBERMAN, S. (1947), "Problem of Combined Action of Carbon Monoxide and Benzene Vapour", *Farmakolt-Toksikol.*, 10: 22; Reference No. 985, page 121, in "Air Pollution", a bibliography by Davenport, S., and Morgis, G. (1954), Bureau of Mines Bulletin No. 357.

COMMITTEE ON AIR POLLUTION (1953), Interim Report, Command 9322, H.M. Stationery Office: 17.

COMMITTEE ON AIR POLLUTION (1954), Report, Command 9322, H.M. Stationery Office: 8.

CRALLEY, L. (1942), "The Effect of Irritant Gases upon the Rate of Ciliary Activity", *J. Indust. Hyg. & Toxicol.*, 24: 193.

DAUTREBANDE, L., and CAPPS, R. (1950), "Studies on Aerosols: Enhancement of Irritating Effects of Various Substances on the Eye, Nose and Throat by Particulate Matter and Liquid Aerosols in Connection with Pollution of the Atmosphere", *Arch. internat. Pharmacodyn.*, 82: 505; abstracted in *Arch. Indust. Hyg.* (1954), 10: 470.

FOULGER, J. H. (1951), "Physiologic Effects of Chemical Contaminants of the Atmosphere", Proceedings of the 1st National Air Pollution Symposium sponsored by Stanford Research Institute: 121.

FRY, J. (1953), "Effects of a Severe Fog on a General Practice", *Lancet*, 1: 235.

HALDANE, J. S. (1931), "Atmospheric Pollution and Fogs", *Brit. M. J.*, 1: 366.

HEMBON, W. C. (1955), "Estimation of Health Hazards from Air Pollution", *Arch. Indust. Health*, 11: 397.

HENDERSON, Y., and HAGGARD, H. W. (1943), "Noxious Gases and the Principles of Respiration Influencing their Action", 2nd Edition, Reinhold, Chapter 9: 131.

LA BELLE, C., LONG, J., and CHRISTOFANO, E. (1955), "Synergistic Effect of Aerosols", *Arch. Indust. Health*, 11: 297.

LAWTHER, P. G. (1955), "Effects of Inhalation of Sulphur Dioxide on Respiration and Pulse Rate in Normal Subjects", *Lancet*, 2: 745.

LEADING ARTICLE (1950), "Synergism in Occupational Exposures", *Indust. Med. & Surg.*, 19: 451.

LEFF, S. (1953), "Social Medicine", Routledge & Kegan Paul, Chapter III; 66.

MINISTRY OF HEALTH, UNITED KINGDOM (1954), "Mortality and Morbidity during the London Fog of December, 1952", Report No. 15, H.M. Stationery Office.

NELSON, N. (1955), "Some Toxicologic Aspects of Atmospheric Pollution", *Am. J. Pub. Health*, 45: 1289.

PEMBERTON, J., and GOLDBERG, C. (1954), "Air Pollution and Bronchitis", *Brit. M. J.*, 2: 567.

REID, D. D. (1956), "Ætiology of Chronic Bronchitis", *Brit. M. J.*, 1: 906.

STANFORD RESEARCH INSTITUTE (1949), "The Smog Problem in Los Angeles County", 2nd Interim Report; Reference No. 631, page 78, in "Air Pollution", a bibliography by Davenport, S., and Morgis, G. (1954), Bureau of Mines Bulletin No. 537.

TAINE, H. (1872), *"Notes sur d'Angleterre"*.

"Threshold Limits for 1956" (1956), *Arch. Indust. Health*, 14: 186.

THE SPASTIC CHILD.[1]

By CLAUDIA BURTON-BRADLEY,

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HAD I chosen the title of this paper myself, I should not have been able to resist choosing "Children with Cerebral Palsy"—that is, the paper should be about children who have a particular disability rather than about a particular kind of child. In so doing, I should have made a plea, not only for the scientific consideration of the afflictions and their causes, but also for the maintenance of dignity in relation to these children as fellow beings.

However, the title was chosen for me. By mutual agreement, it was decided that Dr. Vines would deal mainly with ætiology and classification and that I should deal mainly with investigation and treatment. The subject is vast, and it is difficult in a short paper to select those aspects which will be of greatest interest to you all. However, in addition to commenting on investigation and treatment, I should like to define clearly the terms "cerebral palsy" and "spastic", and to present briefly the classification which I have found most useful from the point of view of clarity and description, and also of its application to investigation and to treatment techniques.

Cerebral palsy, frequently loosely referred to as "spastic paralysis" or "Little's disease", is a term used to cover a group of closely allied, crippling conditions in which there is defective motor function or control. This, as the name implies, has its origin in abnormalities of the motor centres of the brain as distinct from palsies of spinal or peripheral origin. It may manifest itself by the presence of muscle hypertonia or hypotonia, defective balance, defective coordination, the presence of involuntary movements or any other motor dysfunction of cerebral (including cerebellar) origin, with possible resultant defects of speech, posture, ambulation, dexterity and physical deformity. It ranks with poliomyelitis in frequency as a cause of crippling.

The American Academy of Cerebral Palsy, a body consisting of medical men and women concerned with the scientific investigation of cerebral palsy, have adopted the following descriptive definition:

> Cerebral palsy is any abnormal alteration of movement or motor function arising from defect, injury or disease of the nervous tissues contained in the cranial cavity.

The mass of physical, mental and sensory symptoms presented by cerebral palsied children falls into five fairly well defined clinical types. Muscle qualities and involuntary motion form the basis of this classification. First, the symptom complex or type, with some subtypes of this, is stated; this is followed by the limbs involved and then the degree of severity.

Types.

The five types are as follows: (i) spasticity; (ii) ataxia; (iii) athetosis; (iv) tremor—(iii) and (iv) are often referred to as the dyskinæsias; (v) rigidity (also sometimes included in the dyskinæsias).

The following terms are used in relation to the limbs involved: (*a*) hemiplegia—ipsilateral upper and lower limb, designated right or left hemiplegia; (*b*) paraplegia—both lower limbs; (*c*) triplegia—three limbs affected; and (*d*) tetraplegia—all four limbs involved. The degree of the condition is arbitrarily classified as mild, moderate or severe.

Spasticity is found to occur as hemiplegia, paraplegia, triplegia and tetraplegia. The term monoplegia is applied to one affected limb. This must occur very rarely. I have not seen true monoplegia. I have seen hemiplegia with the leg or the arm apparently unaffected; but on careful examination of the other ipsilateral limb at least altered reflexes have been elicited. Spastic triplegia occurs as spasticity in both lower limbs and the right or left upper limb, and is designated right or left triplegia, "right" or "left" being applied to the arm affected.

Ataxia involves all four limbs in the cerebellar type.

Athetosis is found to occur in all four limbs (athetoid tetraplegia), and also as hemiplegia (athetosis in the ipsilateral upper and lower limb—that is, right or left hemi-athetosis). This condition occurs infrequently in the athetoid group, but is a clearly defined clinical entity.

Rigidity is found to occur as tetraplegia, hemiplegia and paraplegia.

The term "diplegia", by virtue of its application sometimes to two limbs and sometimes to affection of the two limbs on both sides of the body, is confusing, and the term "double hemiplegia" appears to me to have no advantage over "tetraplegia" or "quadriplegia".

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on November 29, 1956.

The Spastic Type.

The spastic type is characterized by the presence of some muscles in which the normal stretch or myotatic reflex is augmented. That is, there is an exaggerated contraction of muscles subjected to stretching or a hyperactive stretch reflex. This is spasticity. The qualities of the spastic muscle are such that there are increased irritability, contractibility and tone, and also increased resistance to manipulation; as gravity acts as a force, the kinæsthetic sense appears over-active, in that with change in the gravitational field, there is contraction of the spastic muscles.

Electromyographic examination shows the spastic muscle to have a lowered threshold, an enlarged reflexogenic area and an augmented response.

Tendon reflexes are hyperactive (the eliciting stimulus is indeed itself a stretching of the spastic muscle), and ankle clonus is elicited from spastic calf muscles, and often patellar clonus from spastic quadriceps. The plantar response is extensor. The muscles most frequently spastic are the adductor and flexor muscles of the upper and lower limbs and the pronators of the forearm.

In the spastic, there also occur flaccid muscles (the so-called flaccidity of cerebral origin) as well as normal muscles. This condition gives rise to the outstanding orthopædic problem of muscle imbalance, for not only does the spastic muscle interfere with the function of its antagonist, but if its antagonist is a normal muscle it weakens it by continued stretching, or if its antagonist is a flaccid muscle, severe deformity is brought about. Analysis of the muscle imbalance must form the basis of all orthopædic procedures, whether in the application of an appliance to aid walking or in any surgical procedures.

Interesting examples of mistakes are the application of a toe-raising spring to patients with a spastic calf; this elicits clonus for the child when walking. Another mistake is the surgical lengthening of the tendo Achilles in a child who has a spastic *tibialis anterior* as well as spastic calf muscles. In this case the foot is almost flat, but as the calf group is the more powerful the foot cannot be moved to less than a right angle. The surgical lengthening of the tendon in this case results in a weakening of the spasticity of the calf muscles and the relative over-action of the spastic *tibialis anterior*, with subsequent calcaneous deformity. The patient then becomes a heel walker with the inability to walk without an appliance limiting dorsiflexion, and if both feet have been treated the inability to walk without such appliances together with crutches. The alternative would have been slow movement because of the spasticity in both the calf group and the *tibialis anterior*, time being taken to get the heel down and to push off—a much more desirable state of affairs for the patient.

Two sub-types of spastic are differentiated: (*a*) spastic spastic, with predominance of spastic muscles; (*b*) aspastic spastic, with predominance of flaccid muscles.

The Ataxic Type.

The ataxic type is characterized by a defect of primary coordination. There is interference with the orientation of the body and extremities in space. There is loss of kinæsthetic sense, and normal irritability of muscles is lessened. Muscle contractility is normal, but tone is diminished and power decreased. Reflexes are diminished or absent.

The sub-types are (*a*) the cerebellar type and (*b*) the eighth nerve type. The ataxic type is the rarest of the five types of cerebral palsy, and the eighth nerve type of ataxia is extremely rare.

The Athetoid Type.

Athetosis is involuntary motion which occurs without a definite pattern, present during waking hours and disappearing during sleep; it is an involuntary attempt to move a joint in a certain way, rather than involuntary contraction of individual muscles. The involuntary motion may also appear as poorly coordinated movement or appear against tension. When volitional motions are attempted, involuntary motion distorts the attempt, and non-coordination is secondary to this. The involuntary motion of athetosis varies from barely perceptible movement to the bizarre and changing postures of the dystonic type.

The application of the terms "choreiform" and "choreoathetoid" varies according to different writers on the subject in differentiating between the more rapid hyperkinetic involuntary movements and those which appear more purposive and less random.

W. M. Phelps, of Baltimore (personal communication), differentiates 12 different clinical types of athetosis. These types are not all mutually exclusive. The most frequently occurring are as follows: (*a*) tremor-like, the most frequent, in which athetosis is mainly flexor-extensor; in this type there is a high-stepping gait; (*b*) rotary, in which the athetosis involves mainly the rotary muscles; (*c*) dystonic, in which changing bizarre postures are the feature (this somewhat resembles *dystonia musculorum deformans*); (*d*) tension type; (*e*) non-tension type. In the two last-mentioned, muscle tension or lack of it is the outstanding characteristic. In the non-tension type the patients have flabby musculature, and the neck and back are weak.

The Tremor Type.

Sometimes involuntary motions are true tremors. The tremor type is characterized by involuntary motion, in which there is reciprocal agonist-antagonist contraction regular in rhythm. Reflexes are usually normal, but may appear increased. Two types are differentiated: (i) intention tremor: tremor takes place only with conscious activity—for example, when the patient is trying to write; (ii) non-intention tremor—tremor which is present all the time.

The Rigidity Type.

Rigidity is characterized by an involuntary sustained state of partial contraction of muscles. On examination of the patient, joints flex or extend slowly like the bending of a lead pipe. Reflexes are usually normal, but an extensor plantar response and occasionally ankle clonus may be obtained. Rigidity is frequently associated with pronounced amentia. Rigidity is differentiated into (i) constant and (ii) intermittent. Constant rigidity is further differentiated into flexor, extensor and post-encephalitic; in the last-mentioned there is usually a tremor element as well.

Investigations.

History.

An ante-natal, natal and post-natal history, and a family history, are important aspects of the investigation. They are important, not only from the research angle, but also because they may provide for the parents the explanation of the cause of the condition. This is most important, not only from the point of view of the possibility that further children may be affected, but also because my experience in this regard leads me to believe that no real explanation of the cause of the condition is psychologically damaging to the parents. With adequate explanation they are freed from individual blame, and also from the soul-destroying hate which they can come to project on to their medical adviser, the obstetrician, each other, and society generally. It is important for them that condemnation or guilt is not laid at anyone's door. In the progressive history, points of importance are the age of acquisition of holding the head erect, reaching out for objects, sitting, crawling, standing and walking if these have been achieved, together with any history of convulsions, and whether a dominant hand has been set. It is important early to recognize disturbance of laterality, for further retardation must be expected if the child fails to set a leading hand. Whether one subscribes to the theory of hemispherical dominance or not, the setting of hand dominance is often followed by the development of speech, improvement in behaviour and concentration, and a raising of estimable intelligence quotient.

Physical Examination.

A full physical examination, including examination of reflexes, measurement of the circumference of the head, investigation of eye motions and tongue motions, and

except in the very young infant, type diagnosis, should be made at this stage. Posture achievements should be checked. Can the child sit alone? Can he take his weight? Does he make reciprocal leg movements when placed on his feet? Are there any contractures or any asymmetry?

X-Ray Examination.

Examination of the hips by X ray is sometimes most revealing, indicating muscle imbalance around the hips, sometimes with secondary subluxation or dislocation. Occasionally hypoplasia of the acetabulum or congenital dislocation of the hips is found.

Electroencephalographic Investigations.

Electroencephalographic investigations are valuable from the point of view of research, as well as their indication of the type of medication most likely to be of value for patients with epileptic complications. At the Spastic Centre during the last three years, investigations of over 1000 children have shown 70% abnormal tracings. This includes all presenting patients. Among in-patients there are approximately 50% normal, 43% abnormal and 7% borderline tracings. Some presenting out-patients would be classified as brain-injured and not cerebral-palsied. Some patients, dysrhythmic at an earlier age, have given normal tracings with further maturation.

Sight and Hearing Investigations.

Investigations of sight and hearing are important when visual defect and/or hearing defect is suspected. Visual defects can occur in all types, but they are more frequent in the spastic from imbalance of eye musculature. The ataxic often has visual defects and frequently nystagmus. The Rh athetoids often have paralysis of upward eye motion, and some have poor accommodation.

Some athetoids are deaf, the Rh type having the greatest loss in the high frequencies. Full audiometric estimation of hearing in these cases is most valuable from the point of view of research, speech therapy and education of the patient as a deaf child.

Evaluation of Intelligence.

Evaluation of intelligence is important, both as research and for the placement of the child in an educational set-up. The motor involvement, possible visual and hearing difficulties and exaggerated emotional reaction on the part of the child often affect the responses. As a result the data obtained from a test of intelligence are not a picture of a mental age level. However, by the appropriate test material and modifications, a score can generally be made which will indicate fairly reliably how efficient a child is likely to be at school, with the possible exceptions of the very young, distractible child and the Rh deaf child.

Progress in school will be determined, as with any other children, by factors such as intelligence, home background, qualities of perseverance *et cetera*. Among cerebral-palsied children are found some whose performance in all their school work is somewhat less than one might expect, and some are specifically weak in one subject, such as reading or number work. This is probably related to specific characteristics which affect their response to instruction. Disturbance of attention by extraneous stimuli, lapses of concentration *et cetera*, may directly affect learning. So, too, may over-consciousness of detail and abnormality in the perception of visual, auditory or other stimuli. Changes of handedness as a result of greater motor involvement on one side or more extensive cortical damage may also be contributing factors.

Treatment.

Drugs.

The use of drugs in the main is symptomatic. Those patients with some form of epileptic phenomena are given the anticonvulsants known best to control the particular type, and this is correlated with electroencephalographic tracings. Sometimes it is found that a child whose tracings are indicative of seizures or behaviour disorder, but who does not have convulsions, or whose behaviour is normal, shows improvement in concentration and general school work with the administration of the drugs appropriate to the electroencephalographic pattern.

Of all the antispasmodic drugs, "Myanesin" (mephenesin) and "Myanesin" in combination with "Seconal" ("Seconesin") are found to be most useful to date. "Myanesin" in the elixir form acts quickly, often within ten minutes, and is very valuable on occasions such as just prior to dental attention. "Seconesin" acts more slowly and is less effective but more lasting, and has the additional quality of allaying apprehension. "Equanil" or "Miltown" (meprobamate) would seem to have a somewhat similar effect.

Operative Measures.

Many orthopædic surgical procedures have been used in the past, in an endeavour to correct deformity. Many tendon lengthenings have not been ultimately successful, owing to failure to maintain the position gained, through lack of consideration of muscle imbalance or incorrect selection of cases. The most useful procedures I find in the spastic type are subcutaneous adductor tenotomy for spastic adduction and internal rotation, and wrist fusion in the position of election by the adolescent or adult; in the athetoid type, occasionally a subcutaneous tenotomy of the semi-tendinosus in a severe case will aid extension of the knee in a control brace.

Many neurosurgical procedures have been tried, and with relatively little success. Stoeffel's neurectomy is sometimes of value in the case in which clonus is so severe that it occurs in all walking positions of the foot. Central surgery has in general proved disappointing; but recently it would appear that help may be afforded patients with severe tremor by the recently introduced operative work of section of the anterior choroidal artery or the chemical ablation of the *globus pallidus*—chemopallidectomy. Both these procedures have brought about the cessation of extreme involuntary movements in Parkinson's disease and *dystonia musculorum deformans*.

Physical Treatment.

Physical treatment of the cerebral-palsied child is really habilitation, the aim being to make the child as self-sufficient as possible. In this, all residual capacities must be developed to their fullest. Deformity must be prevented, ambulation acquired, hands used as effectively as possible, speech made intelligible, character and personality developed, and the best possible education for the child's intellectual ability obtained. The two last-mentioned aspects are the most important. As in rehabilitation, treatment is divided into sections.

The acquisition of intelligible speech for many is a great problem. Spasticity of the tongue and facial musculature, or athetosis of the tongue and facial musculature with or without a hearing loss, frequently faces the speech therapist.

In developing the effective use of the hands, the occupational therapist may need to start with the assessment of which is the child's dominant hand, and after this, early activities must seek to obtain the simple fundamentals of hand use—stretch, grasp, release, placement and pinch. Often the therapist must devise methods for overcoming a persistent tonic neck reflex during activity. The teaching of dressing and other self-help skills, of the early stages of writing and, for those patients who cannot achieve relatively fast legible writing, of typing, fall to her lot.

The principles laid down in physiotherapy for the different types of cerebral palsy must be understood by both the occupational therapist and speech therapist. Aims, and goals achieved, must be carried over and maintained in each sphere of treatment, and as far as possible, in the schoolroom and home. The attaining of independent ambulation is the main goal for the physiotherapist. First, there must be type diagnosis and physical assessment, the immediate aims being laid down and long-term goals kept in mind. Relaxation and exercise with the attainment of posture goals form the basis of treatment, with particular

modifications for the spastic, athetoid, ataxic, tremor and rigidity types. A muscle chart is of great value in the spastic type. The usual modalities are used, depending on the stage at which the child is, and on his individual needs. There tends to be a balance defect in most cases even when there is no ataxia. The factors of general muscle weakness, spasticity, involuntary motion *et cetera* contribute to this. The child's normal progression of sitting balance, crawling, pulling himself up, standing and walking is usually delayed, and the picture is one of retarded development. The stages of sitting balance, standing balance and walking reciprocation must be achieved in progression before walking.

Certain braces, calipers *et cetera* and special equipment along with the general physiotherapy programme are of great value. A functional chair, which gives support to back and arms and prevents the child from slipping forward, is important for everyday use and helps to strengthen neck and trunk. At the same time, the beginning of sitting alone is practised on the plinth.

In those cases in which reciprocation is interfered with, training in this is begun early, first on the plinth and later in parallel bars with slide-along grips to prevent the hands from having to be lifted from the bars. Reciprocal skis are also used. A piece of equipment called a reciprocator is used for those children who have no idea of reciprocation.

In teaching standing balance, braces and standing tables are used. The standing table allows the child to practise standing for long periods with a feeling of security while going on with occupational therapy or early school work. This position also helps to develop head control and to strengthen the neck and trunk. When the child is sufficiently advanced, trunk control unsupported must be attained. For this a stabilizer is used. This gives secure support to the legs and feet and leaves the trunk free. The child can make voluntary attempts to control his body, and forward and lateral trunk bending and raising exercises can be carried out. This position allows also for practice of coordinated movements of the upper limbs in such activities as ball throwing and catching. This is of value in preparation for crutch walking. From the stabilizer, for those children who have sufficient hand control, the progression is to parallel bars and standing with crutches, walking with crutches, walking with sticks, and then walking alone.

For those children who do not achieve sufficient hand control (many athetoids and some spastic tetraplegics) progression is from the stabilizer to stabilizer skis. These are skis with legs and feet supported. In these, the child learns, while controlling the trunk, to transfer his weight from one leg to the other in attempted walking. From stabilizer skis, the child progresses to small skis and then to walking alone.

CEREBRAL PALSY.

By W. D. Cunningham,

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No survey of the incidence of cerebral palsy in Australia, or research into its causes has been carried out, mainly because of lack of money to do it well. All available funds are spent in investigation and treatment of the presenting patients at the spastic centres. Dr. Claudia Burton-Bradley, Medical Director of The Spastic Centre, Mosman, has kindly given me the following facts.

At the Centre there are 142 cerebral-palsied children attending each day, and approximately 800 attending as out-patients three to four times a year. New patients are received at the rate of two per week; some of these are pure aments without any evidence of cerebral palsy. Of the latter, however, some have a birth history and electroencephalographic findings suggestive of brain injury.

Dr. W. M. Phelps, of Baltimore, United States of America, estimates that cerebral palsy occurs in seven cases per year per 100,000 of the population, and of these seven children, five survive the neonatal period. As the cultures and medical services of both countries are similar, it is suggested that in all probability the same figures would apply in Australia. If we take the population estimates at June 30, 1956, as given by the Government Statistician (Australia, 9,427,558; New South Wales, 3,553,432—Sydney and suburbs, 1,935,880), the figures for those surviving could be as follows:

Australia	7520 cerebral-palsied patients
New South Wales ..	2880 cerebral-palsied patients
Sydney	1520 cerebral-palsied patients

The last traced patient among the 60 spastic babies who had been delivered at King George V Hospital was born in 1954. From May, 1941, to June, 1954, 46,015 babies have been born in this hospital. This gives an incidence of 0·01 per 1000 live births.

T. T. S. Ingram (1955), in his Edinburgh survey, quotes the incidence as between 2·0 and 2·5 cases per 1000 children.

While the incidence is very low at our hospital, yet a survey of the whole of New South Wales may reveal an incidence comparable with that found in the Edinburgh survey.

My investigation was undertaken in the hope of discovering some new line of thought for research into the causes of infantile cerebral palsy. The affected 52 children studied were born at King George V Hospital and later investigated at The Spastic Centre, Mosman. An additional eight, whose records are unfortunately deficient or absent, are excluded. Cerebral palsy is a descriptive term applying to a group of motor disorders of young children in whom full function of one or more limbs is prevented by paresis, involuntary movement or non-coordination. The 52 children studied were suffering from the different types of cerebral palsy shown in Table I.

Table I.

Type of Palsy.	Full-Time Babies.	Premature Babies.	Total.
Hemiplegia	9	2	11
Tetraplegia :			
(a) Athetoid ..	10	1	11
(b) Spastic	7	1	8
Athetosis :			
(a) With tremor ..	4	0	4
(b) With tension ..	2	2	4
Rigidity	4	0	4
Cerebral agenesis	2	2	4
Amentia	1	1	2
Hypotonia	1	0	1
Mental retardation ..	1	0	1
Speech defect	1	0	1
Tremor	1	0	1
Total	43	9	52

Ætiology.

The analysis of the obstetrical records in these 52 cases showed that the cerebral palsy was not related to race, age, parity, syphilis, virus infection in the mother, type of pelvis or duration of labour.

Rh immunization played a minor but damaging part.

The following other antenatal causes were considered.

Hereditary Factors.

An hereditary ætiology could not be applied to any of the cases.

Diabetes.

There was one mother who suffered from diabetes. The baby developed right spastic hemiplegia. The labour was normal.

Toxæmia of Pregnancy.

There were three cases of toxæmia of pregnancy. One case was mild and the baby was afflicted with rigidity. Labour was induced at the thirty-eighth week. The two others were cases of preeclampsia. In the first of the two, twins were born. The first twin is normal, but the second twin became an ament. Both were delivered by forceps. The weight of the first twin was three pounds four ounces, and that of the second twin three pounds thirteen ounces. The second of the two cases was one of premature labour. The baby was apparently normal for a week after birth, but developed right spastic hemiplegia in infancy. The weight at birth was four pounds fifteen ounces.

Accidental Hæmorrhage.

There were three cases of accidental hæmorrhage. In the first the child is a microcephalic ament. To stop heavy vaginal hæmorrhage during labour, Willett forceps were applied to the baby's scalp and a one-pound weight traction was kept up for one hour. The second baby was delivered after a normal labour, to become a spastic tetraplegic. The third was a premature baby weighing two pounds fourteen ounces, that lived to suffer from athetoid tetraplegia.

Rh Immunization.

Rh immunization with jaundice and kernicterus developing in neonatal life occurred in four cases. None of these babies was premature, and all had exchange transfusions. Three of the babies became athetoid tetraplegics, and the fourth mildly athetoid.

Type of Delivery.

There were 18 cases in which the type of delivery could have caused cerebral trauma; of these, one was a breech delivery and 12 were mid-forceps deliveries; three other babies were delivered after a prolonged second stage (over three hours), and two after a precipitate labour.

Delivery Without Mechanical Trauma.

Thirty-four infants were delivered without mechanical trauma, 32 after a normal labour and two by Cæsarean section.

The indications for Cæsarean section were as follows. The first patient had a history of three previous neonatal deaths; the causes were unknown. The present baby, weighing seven pounds fifteen ounces, was of Class C (see below) after delivery, and later developed spastic hemiplegia. In the second case the operation was performed for fœtal distress from an unknown cause during the first stage of labour. The baby weighed eight pounds eight ounces and was of Class B at birth. Signs of cerebral irritation, a whining cry and head retraction developed, and the baby subsequently became a spastic hemiplegic.

Comment.

Records are kept of all babies at birth in regard to their breathing and general condition according to the following classes:

Class A: Breathed immediately.

Class B: Condition good but not excellent—recovered within five minutes.

Class C: Condition fair to poor, moderate anoxia. Respiration first absent or gasping. Active resuscitation needed and child responded. The condition became good within thirty minutes.

Class D: Condition serious (severe anoxia or collapse). Respirations absent at first. Skin pale and clammy. Muscle tone poor. Heart feeble and irregular. Slow response to active resuscitation, and condition still not good thirty minutes after birth.

Table II sets out the condition of the babies at birth according to the foregoing classification.

It is evident that 19 of the babies were in poor condition at birth, requiring active resuscitation.

Despite the small number of the series, it is significant that anoxia at birth is a factor.

In the neonatal period the babies were also handicapped. Many had attacks of cyanosis and cerebral irritability, and feeding difficulties were common. These details are set out in Table III.

One baby, showing symptoms of cerebral irritation, had convulsive seizures.

Special Investigations Performed.

Two of the babies with signs and symptoms of cerebral irritation had subdural hæmatomata that were successfully tapped, with some relief of symptoms. Unfortunately one developed right-spastic hemiplegia and the other mild spastic tetraplegia.

TABLE II.

Condition at Birth.	Premature Babies.	Full-Term Babies.	Total.
Class A	1	10	11
Class B	3	19	22
Class C	5	12	17
Class D	—	2	2
Total ..	9	43	52

One infant, who subsequently developed "rigidity", was found on electroencephalographic examination to have left-sided brain damage, and this, together with the symptoms of convulsions, was the indication for tapping for subdural hæmatoma; the procedure was unsuccessful.

Three lumbar punctures were performed; one was described as "dry"; another produced blood-stained cerebrospinal fluid; and the last produced xanthochromic cerebrospinal fluid containing 338 red blood cells per cubic millimetre.

TABLE III.

Type of Affection.	Premature Babies.	Full-Term Babies.	Total.
Cyanotic attacks alone ..	6	8	14
Cerebral irritation ..	—	8	8
Cyanotic attacks and cerebral irritation ..	—	3	3

Discussion.

The very title of Little's paper of 1862 (Ingram, 1955) implies an interpretation of the causes of cerebral palsy: "On the Influence of Abnormal Parturition, Difficult Labour, Premature Birth and Asphyxia Neonatorum on the Mental and Physical Condition of the Child, Especially in Relation to Deformities". If we could add "Anoxia in Utero and Hæmolytic Diseases of the Newborn" to Little's imposing title, all causes would seem to be covered.

Although there were only three proven cases of accidental hæmorrhage, it is my clinical impression that the majority of premature labours are initiated by some accidental hæmorrhage. In seven of the nine cases of premature labour it was noted that labour had coincided with a "show" of blood. Anoxia from partial separation of the placenta must then wreak cerebral damage. Eastman and De Leon (1956) are of the same opinion.

It would appear, from this small series of cases, that in the majority the brain damage is done before birth, as demonstrated in the two cases in which delivery was by Cæsarean section. Was anoxia during induction of anæsthesia the cause in these two cases? In 32 cases labour was apparently normal. Therefore one can conclude that mechanical trauma at birth is a small factor in present-day obstetrics.

In babies who had prolonged neonatal jaundice (kernicterus) due to the Rh immunization factor a similar clinical picture seemed to develop. Kernicterus is now

recognized as dangerous to the small premature baby, and this, too, may well cause cerebral palsy. With repeated blood transfusions given to babies suffering from hæmolytic diseases of the newborn, the figures for cerebral palsy from this cause should fall.

On clinical findings, except in the cases of Rh immunization, correlation of the ætiology with the various forms of cerebral palsy was not possible; but perinatal anoxia inflicting varying degrees of cerebral damage would appear to be responsible for the different neurological sequelæ.

In six cases in this series there were no antenatal and intranatal complications, and the babies appeared to be perfectly normal at birth. Why they should have developed a cerebral palsy is problematical. Possibly an unnoticed phase of fœtal anoxia may have been passed through during labour, with the production of a subependymal hæmorrhage.

Eastman and de Leon (1956), who investigated 96 cases of cerebral palsy, stated that more data on the obstetrical ætiology were needed, and urged the active help of all obstetricians throughout America. These sentiments are true for Australia also.

While there is great enthusiasm, and a vast amount of money is being expended in bricks and mortar, to provide the splendid treatment for cerebral palsy in Australia, yet there is a great need for plodding investigation and research for its prevention.

With better understanding of antecedent obstetrical factors, the obstetrician may be forewarned of possible neurological damage, and may enlist the pædiatrician's aid promptly for the newborn.

Improved technique in delivery and the earlier recognition of maternal complications producing anoxia must result in a lessening of the incidence of cerebral palsy.

Acknowledgements.

I should like to thank Dr. Claudia Burton-Bradley, Miss M. Marshall, of the Records Department of King George V Hospital, and the Sydney University Research Committee, for their cooperation.

References.

EASTMAN, J., and DE LEON, M. (1956), "The Etiology of Cerebral Palsy", *Am. J. Obst. & Gynec.*, 69: 955.

INGRAM, T. T. S. (1955), "A Study of Cerebral Palsy in the Childhood Population of Edinburgh", *Arch. Dis. Childhood.*, 30: 150.

LATHAM, D., ANDERSON, G., and EASTMAN, N. J. (1954), "Obstetrical Factors in the Etiology of Cerebral Palsy", *Am. J. Obst. & Gynec.*, 68: 96.

UNIVERSAL FINGER SPLINT.

By JOHN NOVOTNY, M.B., B.S.,

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THE use of an aluminium splint is now a generally accepted method for immobilization of an infected or injured finger. At Saint Vincent's Hospital, Sydney, we have been using the following two types of aluminium splint: one for the thumb, designed in this hospital by Dr. John Tonkin several years ago and generally known as Tonkin's splint; the other for the fingers and known as T-splint. However, the construction of the latter splint confronted us with considerable technical difficulties, since the cutting of the T-splint in one piece means wasting about 80% of the material used, and in addition each splint has to be cut especially for the particular finger. The wasting of both material and time in constructing each individual T-splint led me to devise a new method of making the splint. This, briefly, consists of cutting the digital arm along the palmar arm and then bending the former over the latter, thus finishing with a T-splint.

The advantages of the new splint over the old one are as follows: (i) It is more than four times as economical. (ii) It is more than twice as quick to make and apply. (iii) It is applicable to any finger of either hand; that is, once the splint is ready, it fits any of the eight fingers, and in the initial stages it may be used as Tonkin's splint for the thumb of either hand. Thus it is truly universal. (iv) It may be easily and economically prefabricated in spare time and stored in the surgeon's drawer, and with the help of a small pair of cutters adjusted to the final requirements of the individual patient at several seconds' notice, since no distinction between the individual fingers is then necessary.

The splint may be used for any condition requiring immobilization of a finger.

Construction.

A standard type, applicable to most cases—that is, an average and large hand—is described here. It may be modified for a smaller hand if required. The splint is made as follows:

1. First cut out a quadrilateral area measuring 4·5 by 26 centimetres (1·9 by 10 inches) from an aluminium sheet one millimetre thick, and draw two lines 1·5 centimetres apart parallel to the long side, using a sharp nail. Draw a line at right angles to the long side, passing through the centre of the latter. A second line is then drawn at an angle of 45° through the centre of this latter line (Figure I).

FIGURE I.

First stage of construction of splint: interrupted line, auxiliary line; solid line, cutting line.

2. Cut along the lines as shown, and bend the two parts along the oblique line (Figure II).

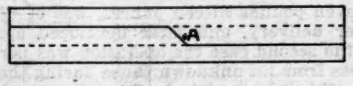

FIGURE II.

Second stage of construction: A, oblique line.

3. Cut along the oblique line so as to join the inner ends of both cuts. This gives two splints. The reason why we cut two splints at the same time is that there is no wasting of aluminium, and secondly, when one splint is cut the other splint is cut automatically (Figure III).

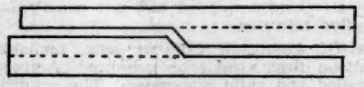

FIGURE III.

Third stage.

4. Take one of the splints and cut along the remaining line towards its centre, leaving 1·8 centimetres uncut, so that two arms are left—the long one the palmar arm, the short one the digital arm (Figure IV).

5. Take the digital arm and bend it over the palmar arm, to cross the transverse axis of the latter and form with it an angle of 75°. This angle has been found to be the most satisfactory. Then hammer slightly the site of bending (Figure V).

6. Now bend the remaining aluminium triangle towards the digital arm at an angle of 90° to the plane of the palmar arm. This can well be done on an iron rail

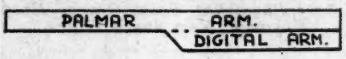

FIGURE IV.
Fourth stage.

possessing a sharp edge. Place the splint on the rail, the digital arm facing downwards and the outer edge of the palmar arm lying parallel with and exactly on the edge of

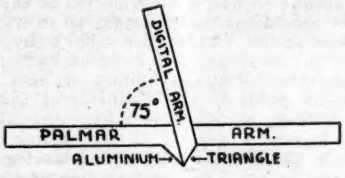

FIGURE V.
Fifth stage.

the rail, so that the aluminium triangle protrudes outwards in the horizontal plane: now hammer the triangle down to 90° (Figure VI).

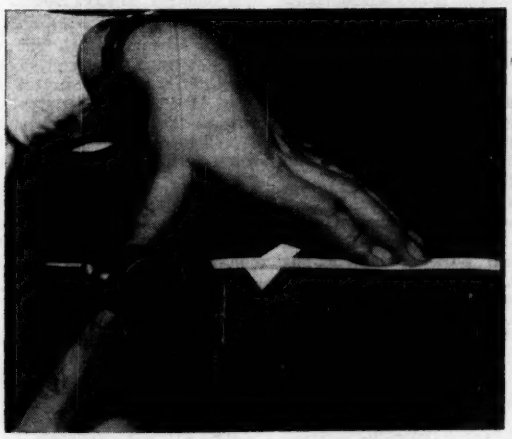

FIGURE VI.
Sixth stage.

7. Now turn the splint to its other face so that the triangle points upwards, and hammer it down to the digital arm so that the latter is firmly gripped between the former and the palmar arm (Figure VII). The tip of the triangle should lie in the longitudinal axis (that is, the mid-line) of the digital arm if the angle between the two arms is correct. An important point to remember is that the triangle should be hammered first from above down and from each side so as to produce its tight and neat apposition with the palmar arm. On no account must an attempt be made to flatten the triangle out by heavy hammering, since it would weaken the joint. The triangle has its important function in lending a support to the digital arm when the splint is applied to the left hand. At this stage the splint may be stored in the surgeon's drawer for use when required.

8. To apply the splint (Figure VIII), place the palmar arm against the palm, the digital arm against the particular finger, and mould the splint as required. Always make sure that the digital arm is sufficiently bent in the form of an arch so that the diseased finger is immobilized in the position of rest. Now adjust the length of the arms as necessary and cut off the protruding angles. When the splint is for the right hand, bend sharply the two halves

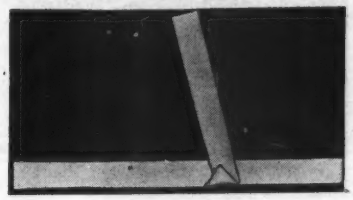

FIGURE VII.
Seventh stage.

of the palmar arm upwards at the triangle and then bend them downwards, so that the triangle sinks into the same plane as the rest of the palmar arm. (See Figure VIII.)

As was mentioned earlier, the splint may be used for any finger of either hand.

FIGURE VIII.
Eighth stage.

9. When the splint is used for the right hand (Figure IXA), the obtuse angle looks to the right and the triangle upwards. When it is used for the left hand, the obtuse angle looks to the left (Figure IXB).

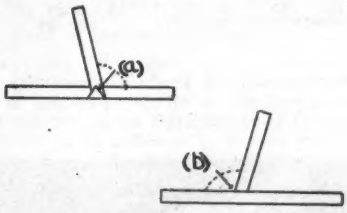

FIGURE IX.
(*a*) Right hand splint; aluminium triangle up. (*b*) Left hand splint; aluminium triangle down.

10. Now apply "Leucoplast", one inch wide, starting at the outer end of the digital arm and bending it over the inner edge of the arm. Then cover similarly the palmar arm. Note that the digital arm should be covered first. This manner of covering gives the digital arm an extra support (Figure X).

11. Now put on the splint again and bridge over the gap between the two ends of the palmar arm on the back of the hand by applying a strip of "Leucoplast" from the radial to the ulnar side (Figure XI). Fix the finger to the digital arm also by a strip of "Leucoplast".

12. The splint at the stage shown in Figure IV may be used as a Tonkin's splint after both the digital and palmar arms have been modified as needed (Figure XII).

13. When the splint has been covered with "Leucoplast" (Figure XIII), the splint for the thumb is also ready.

Summary.

The new splint, in addition to being more than four times as economical as the old one, is also twice as quickly made and applied, and thus greatly helps towards the smooth running of a busy casualty department or private practice. Furthermore, it is truly universal, since it is applicable to any finger of either hand and in the initial stages may be used as Tonkin's splint for the thumb.

FIGURE X.
The splint covered with "Leucoplast".

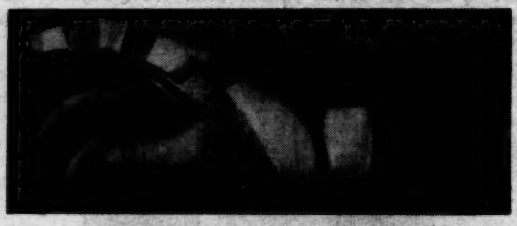

FIGURE XI.
The right hand splint applied.

FIGURE XII.
The splint in the fourth stage of construction prepared for use as a Tonkin's splint.

FIGURE XIII.
The splint applied to the thumb.

Acknowledgements.

I wish to express my thanks to the Sisters of Charity of Saint Vincent de Paul for the photographs and for typing this article. I wish also to thank the honorary surgeons of Saint Vincent's Hospital, Dr. D. P. Rowe, Dr. Hugh Smith and Dr. A. F. Dwyer, for their encouragement. In particular I wish to acknowledge my gratitude to Dr. Noel Curtis Newton for having kindly revised this paper.

MORNING STIFFNESS IN RHEUMATOID ARTHRITIS.

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DURING investigations (history taking) of patients suffering from rheumatoid arthritis, I have been struck again and again by the number who, partly voluntarily and partly on direct questioning, have admitted to the presence of a condition which can best be termed morning stiffness.

Because this symptom has kept recurring in the history taking of rheumatoid arthritis, I have started to give it the consideration it deserves. I have searched the literature and looked around to find a confirmation of this symptom, which I have considered so important as to warrant more detailed investigation. There are not many papers on this subject; but there are a few which have fully borne out the importance of this symptom. I have considered it a very useful guide in the diagnosis of this condition and of the stages, as it were, which the disease goes through; it is also a pointer to the degree of activity taking place in the affected joints. Considering the importance of this symptom, I was rather amazed that so little has been written about morning stiffness in the medical literature. Not only will it confirm that the patient is suffering from rheumatoid arthritis in the majority of cases when this symptom is present, but it also tells us about the degree of activity—whether the disease is active or quiescent, or at a later stage whether it has been reactivated. Being such a reliable symptom, it certainly deserves to be emphasized, and I have always found it a useful symptom in assessing my cases.

Before I describe four cases in some detail, let us be clear in our minds what is meant by morning stiffness. The term was first employed by Cobb and Warren (1954) and later by Professor Lansbury (1956). I am naturally open to correction, but as far as I know, after some searching, these are the only references to the subject. It is described as a numbness or weakness which wears off during the day.

The definition of stiffness is "not working freely, sticking, offering resistance to a changing position, unbending, lacking suppleness, hard to move". The important thing about morning stiffness is that *pari passu* with the patient's activity it wears off as the day progresses, and even in the most severe cases it is much less in two or three hours. Apart from gradually increasing activity, it is well known to most patients that aspirin in some form or other is a great help in shortening the duration of morning stiffness. All patients suffering from rheumatoid arthritis have at least questionable morning stiffness. It is a symptom of far greater importance than has been generally realized.

Simple inquiry about it in many cases, or in other conditions known to be associated with morning stiffness but with no other signs of arthritis, means, if it is found, that the patient should be kept under observation, with the thought in mind that he or she may eventually develop rheumatoid arthritis.

The condition occurs after a night's rest and is noticed by the patient on awakening. Of course, the same symptom may occur in affected joints and muscles after any rest of some duration; but here the stiffness will not be of the same degree as after a full night's rest. It is "morning stiffness in miniature", and comes on after the patient has been sitting or lying down for a time.

It is a manifestation of muscular and joint disease from a wide range of noxious agents—for example, the trauma of direct blows, acute non-articular rheumatism such as fibrositis, and also osteoarthritis, especially in the spine, hip and knee. It is a well-known symptom after a day in the saddle or an occasional game of bowls or tennis or any unaccustomed exercise ("With long travel I am stiff and weary."—*A Comedy of Errors*, 1, 2, 15).

Symptomatically, morning stiffness is the hallmark of disease of the locomotor system. Therefore, although it is

not exclusively confined to rheumatoid arthritis, it can be seen in this condition with such regularity that one could say without exaggeration that it is a constant symptom. It is almost a *sine qua non* in rheumatoid arthritis.

Whenever the result of exercise it will mostly affect the muscles and joints involved in the exercise. This type of stiffness may be very widespread and will wear off in a comparatively short time—as a rule within a few days. On the other hand, it may aggravate the stiffness which is the result of rheumatoid arthritis. In my experience the morning stiffness, all things being equal, lasts longer in older patients. ("Well, corresponding to your stiff age."—*Cymbeline*, III, 3, 32.) In this regard I cannot agree with Professor Lansbury (1956*b*), who states that it appears to be unrelated to age. In my experience age does play an appreciable part in the degree and duration of morning stiffness.

Many patients will not volunteer information regarding the stiffness. I definitely advise that in cases of suspected rheumatoid arthritis this important symptom should be thought of and every effort made to elicit the relevant information. It is surprising how often the patient will withhold this information, which is a valuable aid to diagnosis.

With regard to the duration of morning stiffness many factors are involved, such as the type of joint or joints, the number of involved joints, the type of disease (acute, subacute or chronic), the degree of disease, the degree of atrophy of muscles, the sensitivity of the patient, *et cetera*. I invariably apply the duration of morning stiffness as a measuring stick of activity of the disease, and attempt to gauge the course of the disease accordingly. Together with other important symptoms, such as muscular weakness, degree of pain on activity and at rest, and the erythrocyte sedimentation rate, I find it a reliable guide in assessing my patients.

Reports of Cases.

CASE I.—Miss P., aged twenty-four years, is suffering from generalized rheumatoid arthritis involving the upper and lower limbs, of nine years' known duration. Her erythrocyte sedimentation rate is 54 millimetres in sixty minutes (Wintrobe). On waking she has to limber up her arms and legs, opens and closes her hands at least 30 times, and loosens up her elbows by constantly flexing them. The The duration of morning stiffness is three and three-quarter hours.

CASE II.—Mr. G., aged thirty-eight years, is suffering from rheumatoid arthritis of six years' duration. The joints involved are the wrists, hands and knees. His erythrocyte sedimentation rate is 40 millimetres in sixty minutes (Wintrobe). He has to have a hot bath before breakfast and can "hardly move" before this. The duration of morning stiffness is three hours.

CASE III.—Mrs. S., aged fifty years, has had rheumatoid arthritis for twelve years, affecting the hips, knees, ankles, wrists and hands. She is "stiff as a board" on awakening and is unable to get out of bed in the usual way; she has to roll out. Her erythrocyte sedimentation rate is 24 millimetres in one hour (Wintrobe). The duration of morning stiffness is four and a half hours.

CASE IV.—Miss R., aged forty years, has had rheumatoid arthritis for fourteen years, affecting the hips, knees, ankles, elbows and hands. She has to take two "Disprin" tablets at 5.30 a.m. before she can move about. Her erythrocyte sedimentation rate is 32 millimetres in one hour (Wintrobe). The duration of morning stiffness is three and three-quarter hours.

Summary.

I have tried to stress the importance of morning stiffness in the diagnosis and assessment of cases of rheumatoid arthritis. Evidence shows it to be a symptom practically invariably present in this disease, and although it is very sparingly mentioned in the literature I think its importance cannot be overstated. One should always try to elicit this information from the patients and give it the consideration it deserves.

Acknowledgements.

I wish to record my thanks to Dr. N. C. Wright, Medical Superintendent, Waterfall Sanitorium, and Dr. Brian Haynes, rheumatologist, Saint Vincent's Hospital, for their help and advice.

Bibliography.

COBB, S., WARREN, J., THOMPSON, D., and CIOCCO, A. (1954), "Epidemiology of Rheumatoid Arthritis with Particular Reference to Importance of Morning Stiffness", *Pennsylvania M. J.*, 57: 37.

HENCH, P. S. (1948), "Rheumatism and Arthritis; Review of American and English Literature of Recent Years (Ninth Rheumatism Review)", *Ann. Int. Med.*, 28: 66.

LANSBURY, J. (1956*a*), "Quantitation of the Activity of Rheumatoid Arthritis: 1. A Method for Recording its Systemic Manifestations", *Am. J. M. Sc.*, 231: 616.

LANSBURY, J. (1956*b*), "Quantitation of the Activity of Rheumatoid Arthritis: 2. Recession of Morning Stiffness as Patients Go into Remission", *Am. J. M. Sc.*, 232: 8.

LANSBURY, J. (1956*c*), "Quantitation of the Activity of Rheumatoid Arthritis: 5. A Method for Summation of the Systemic Indices of Rheumatoid Activity", *Am. J. M. Sc.*, 232: 300.

LANSBURY, J., and HAUT, D. D. (1956), "Quantitation of the Manifestations of Rheumatoid Arthritis: 4. Area of Joint Surfaces as an Index to Total Joint Inflammation and Deformity", *Am. J. M. Sc.*, 232: 150.

ROPER, M. W., and BAUER, W. (1945), "Rheumatoid Arthritis: Its Varied Clinical Manifestations", *New England J. Med.*, 233: 592.

STEINBROCKER, O., TRAEGER, C. H., and BATTERMAN, R. C. (1949), "Therapeutic Criteria in Rheumatoid Arthritis", *J.A.M.A.*, 140: 659.

WOOLSEY, T. D. (1952), "Prevalence of Arthritis and Rheumatism in United States", *Pub. Health Rep.*, 67: 505.

HUMAN SALMONELLOSIS IN VICTORIA.

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SINCE 1944 a number of reports have been published concerning the incidence of salmonellosis in Australia by Atkinson and her colleagues (Atkinson *et alii*, 1944, 1947, 1952, 1953), and in Queensland by Mackerras and Mackerras (1949). The results recorded in these papers have provided information on the occurrence of Salmonella serotypes (or species) not only in humans but also in a number of domestic and native animals. To our knowledge, except for a report on an outbreak of gastro-enteritis by Mushin (1948), no information regarding the incidence of Salmonellæ in humans in Victoria has been recorded in the past eight or ten years.

For some years the Public Health Laboratory in Melbourne has undertaken the identification of most Salmonella serotypes (or species) isolated in Victoria. In view of the importance of Salmonella infections in the community, it is the intention of this paper to record the isolations of these organisms and to indicate certain features which may be of significance in an epidemiological approach to the subject. There can be no doubt that the number of isolations of the organisms represents only a small proportion of Salmonella infections, mainly because bacteriological investigation of patients with gastro-enteritis and "mild stomach upsets" and of their contacts is not sought as a routine procedure by the medical practitioner; if the service offered by the Public Health Laboratory was used more widely in such cases, it is certain that valuable information regarding the spread of these infections could be obtained.

Origin of Strains.

Most strains of Salmonella were isolated in the Public Health Laboratory from fæcal specimens of children and adults suffering from gastro-intestinal infections, while many of the remainder were isolated from patients at the Royal Children's Hospital in Melbourne and were forwarded to this laboratory for identification. The remaining strains were received from several of the larger public hospitals in Melbourne, and occasionally from country hospitals and laboratories. Over 90% of all the Salmonellæ recorded were isolated from people living in the urban and suburban districts of Melbourne; only occasionally during the period

of investigation were specimens or organisms received from the outlying country areas of Victoria.

All organisms were identified by methods similar to those described by Edwards and Bruner (1942) and more recently by Cooper (1956); when the need arose, organisms were submitted to Dr. Joan Taylor of the Central Public Health Laboratory, Colindale, England, for either confirmation or, alternatively, full antigenic analysis.

Results and Discussion.

Table I indicates the numbers of each species of Salmonella isolated each year during the six-year period from January, 1950, to December, 1955, together with the total number of organisms isolated in that period. As may be seen from these results, the number of Salmonellæ isolated each year has increased, and approximately three times as many isolations were made in 1955 as in 1950. This does not necessarily represent an increase in the incidence of these organisms over the past six years, but rather is it a reflection of the increasing number of specimens examined each year, particularly during the past two or three years. The results have not only assisted in the treatment of the individual patients, but also have been useful in studying the epidemiology of the infection.

Twenty-nine different species of Salmonella were isolated during the period under investigation. Of these, two were new species (*Salm. victoria*[1] and an unnamed species *Salm.* sp. 1,6,14,25:gm), one isolation of each type being made. These organisms were antigenically analysed by Dr. Joan Taylor, and each was confirmed as a new species. Although the possible contacts of the patients were investigated, no clue could be obtained as to the origin of the strains.

When the incidence of the various Salmonella species is discussed, they may best be referred to the "O" (somatic) antigenic groups of the Kauffman-White schema.

[1] Antigenic formula: 1,9,12 : 1w,1·5.

Group B Salmonellæ.

Salm. typhi-murium.—As has been found in practically every country, *Salm. typhi-murium* is by far the most frequent cause of gastro-enteritis in the community; 80% of all Salmonellæ isolated in this State have been strains of this organism. While a few of the isolations were made in cases associated with food poisoning outbreaks, the majority of strains were derived from children (and, less often, adults) suffering from gastro-intestinal upsets for which no epidemiological explanation could be determined. Reference to the graph (Figure I) indicates that there is a seasonal fluctuation of the infections; the significance of this will be discussed later. While some of the cases could be attributed to direct familial contact, the majority appear to be sporadic.

Salm. derby.—In 1948 Mushin reported a large outbreak of gastro-enteritis caused by *Salm. derby* which occurred in a Melbourne hospital; 68 patients were found to be excreting this organism. However, since 1950 only 18 strains have been isolated from patients; it appears from the figures recorded by Atkinson *et alii* that this organism is more common in other States of Australia than in Victoria. The cases we have examined have been completely unassociated; it is possible that the organism is endemic in the community, but that its pathogenicity for the healthy person is not great.

Salm. reading.—No record exists of *Salm. reading* occurring in Victoria prior to 1955, although several strains from other States were recorded by Atkinson *et alii* in 1944. The 12 strains isolated here were derived from cases connected with a food poisoning outbreak subsequently traced to a Continental-type sausage containing raw pork. As far as could be ascertained, no human carrier of the organism could be held responsible for the contamination of the sausage, and it seemed possible that the organism was derived from the pig meat. As yet, however, no *Salm. reading* has been isolated from a number of pigs that have been examined.

TABLE I.

Salmonella Isolations in Victoria, 1950 to 1955.

"O" Antigenic Group.	Salmonella Species.	Number Isolated in						Total Number Isolated.
		1950	1951	1952	1953	1954	1955	
B	*Salm. typhi-murium*	46	104	177	139	262	260	988
	Salm. derby	1	1	3	8	2	3	18
	Salm. paratyphi B	—	1	1	—	—	1	3
	Salm. reading	—	—	—	—	—	12	12
	Salm. chester	—	—	—	1	—	—	1
	Salm. stanley	—	—	—	—	1	—	1
C_1	*Salm. choleræ-suis* var. *Kunzendorf*	—	—	2	1	2	2	7
	Salm. potsdam	—	—	—	3	—	—	3
	Salm. singapore	—	—	—	2	—	—	2
	Salm. oranienberg	—	—	1	—	—	—	1
	Salm. bareilly	1	—	—	—	—	—	1
C_2	*Salm. bovis-morbificans*	8	1	10	10	18	17	59
	Salm. newport	—	9	1	—	8	16	34
	Salm. muenchen	—	—	—	—	1	—	1
	Salm. oregon	—	—	—	1	—	—	1
D	*Salm. enteritidis*	2	—	—	1	—	2	5
	Salm. victoria	—	—	—	—	1	—	1
E_1	*Salm. anatum*	2	3	1	1	4	2	13
	Salm. orion	—	2	2	3	—	—	7
	Salm. london	—	—	2	—	—	—	2
	Salm. meleagridis	—	—	—	1	—	—	1
	Salm. give	—	—	—	—	—	1	1
E_2	*Salm. cambridge*	—	1	—	—	—	—	1
	Salm. newington	—	—	—	1	—	—	1
E_4	*Salm. senftenberg*	—	2	15	16	—	—	33
G	*Salm. worthington*	1	—	—	1	—	—	2
I	*Salm. orientalis*	—	—	—	—	—	3	3
Others	*Salm. adelaide*	3	6	10	7	12	9	47
	Salm. sp. 1, 6, 14, 25, gm.	—	—	—	—	1	—	1
Total yearly isolations		59	130	225	196	312	328	1250

Salm. paratyphi B, *Salm. chester*, *Salm. derby*.—*Salm. paratyphi B*, *Salm. chester* and *Salm. derby* have no significance in the general pattern of salmonellosis in this State, only isolated cases being recorded. Their origin is obscure except for two cases of paratyphoid fever which were almost certainly acquired in, or on transit from, Europe.

Group C Salmonellæ.

Group C Salmonellæ are relatively unimportant. *Salm. choleræ-suis* var. *Kunzendorf*, although commonly found in animals (particularly pigs), has been isolated from human patients only seven times in the past six years. Single cases of *Salm. barielly* and *Salm. oranienberg* have been obtained; these organisms were introduced into England during the war in American spray-dried egg (Medical Research Council, 1947), and have since become endemic in that country. These strains have occasionally been noted in Australia, more often in animals than in man; the origin of these cases is obscure. The isolation of *Salm. potsdam* from several patients can be directly attributed to a batch of infected desiccated coconut from which it was subsequently isolated (Wilson and Mackenzie, 1955). *Salm. singapore* has been isolated on two occasions in the past six years and does not appear to be endemic in the population.

Group C_1 Salmonellæ.

The organisms in this group C_1 are of next importance to *Salm. typhi-murium* in this State.

Salm. bovis-morbificans. — Until 1952 very few strains of *Salm. bovis - morbificans* were recovered from humans in Victoria. Since then, however, the numbers have increased, and the isolations seem to follow the regular seasonal fluctuation observed with *Salm. typhi-murium*. This species was responsible for a large outbreak of infantile gastro-enteritis in Brisbane (Mackerras and Mackerras, 1949); it was also implicated in a major outbreak of food poisoning caused by infected meat pies in England (Miller, Nicol and Ramsden, 1955). It would seem from these two reports that this organism has a relatively high pathogenicity for man, and it obviously has become endemic in the Victorian population over the last four years.

Salm. newport.—Apart from an isolated outbreak of gastro-enteritis in a Melbourne hospital in 1951, *Salm. newport* has been of little consequence in this State until the last two years. Since then the number of isolations has steadily increased, and it now appears to be firmly established as an endemic strain. It is commonly found in Europe, and it may be that the migrant population has been responsible for its more widespread existence in this community. The organism has been isolated on occasions from Continental-type sausages, and because of their increasing popularity these may constitute at least a substantial source of infection. There is no doubt that if the trend continues this species will be of considerable importance in human infections in this State.

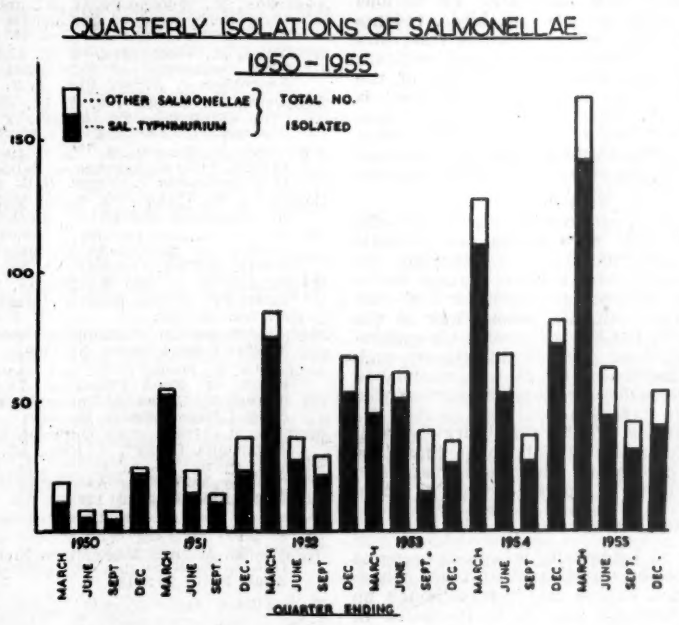

FIGURE I.

Quarterly isolations of Salmonellæ, 1950 to 1955.

Group D Salmonellæ (excluding Salm. typhi).

Group D Salmonellæ are of little importance. *Salm. enteritidis* has occasionally been associated with human infections. Surprisingly enough, this organism was found frequently in rats living in close proximity to the human population. There is ample evidence in the literature to indicate that certain strains of this species are highly pathogenic for man, and this perhaps suggests that these rodents do not constitute a major source of Salmonella infection in this community.

Group E Salmonellæ.

Salm. anatum.—Only sporadic cases of infection due to *Salm. anatum* have occurred each year, and it appears to have little general importance.

Salm. senftenberg.—*Salm. senftenberg* was isolated frequently during 1952 and 1953; since then it has apparently completely disappeared. Almost certainly it was established here by the infected desiccated coconut held responsible for the outbreak of typhoid fever in 1953 (Wilson and Mackenzie, 1955). On numerous occasions it was isolated from samples of coconut, and practically all patients (and carriers) excreting this organism admitted that they had consumed the material in one form or another. It is notable that a number of people were symptomless excretors, and it would seem that this organism has a low pathogenicity for man. This is borne out by the fact that since the prohibition of the coconut it has disappeared, which suggests that it has not been established endemically in the population.

Salm. london, *Salm. meleagridis*, *Salm. newington.*—The few infections by *Salm. london*, *Salm. meleagridis* and *Salm. newington* were recorded during the period referred to above, though they could not be attributed to the infected coconut; none have been isolated since that period.

Other Salmonellæ.

Salm. orientalis.—Atkinson *et alii* first reported the isolation of *Salm. orientalis* in Australia in 1953; their strain was obtained from an infected calf in Queensland. The organism was not found in Victoria until early in 1955, when three strains were isolated from children with gastro-enteritis. The first patient lived in a dairying community and was admitted to a hospital in Melbourne; the two remaining children appeared to have been infected in the hospital. No further cases have occurred since that period.

Salm. adelaide.—*Salm. adelaide* is the only other Salmonella type which is endemic in the population; sporadic cases have been reported throughout the past six years. Most patients were young children with mild to moderate symptoms of gastro-enteritis which responded readily to antibiotic therapy. Early in 1955 several cases occurred in an infant welfare institution, but in spite of careful investigation it was not possible to establish any definite focus of infection.

Discussion.

Figure I illustrates a significant feature of the incidence of salmonellosis in this State. It is evident that the infections (influenced chiefly by those due to *Salm. typhi-murium*) exhibit a seasonal fluctuation throughout the year. The maximum number of isolations are obtained in the first quarter of the year, and in fact, between 40% and 50% of the total yearly isolations are recorded in this period; thereafter a decrease in infections occurs, reaching a minimum in the third quarter and subsequently increasing during the final quarter of the year. This periodic fluctuation suggests a climatic influence on the incidence of salmonellosis, and as is generally realized, the warm to hot months from October to March are more favourable for the transmission of the organisms than are the cooler late autumn, winter and spring months.

In any epidemiological approach to the subject of Salmonella infections in the human population, the reservoirs of the organism must primarily be considered. Many sources of infection have been implicated by various authors; human agents, domestic animals, birds, rodents and insects (particularly flies) are all possible reservoirs and disseminators of these organisms. No good evidence exists to incriminate one agent to the exclusion of the others, but certainly during the summer months there is a pronounced increase in the insect population, and transmission of the organisms by flies, particularly in unsewered areas, is possible. But it seems unlikely that the increase in incidence of Salmonella infections during the summer period is entirely due to these agents.

While a few outbreaks of gastro-enteritis may be directly associated with a single focus of infection, the sporadic cases are by far the most numerous and constitute the major problem of salmonellosis, at least in this State. Whatever the primary source or reservoir of the Salmonellæ may be, in our opinion a second link in the chain of infection—namely, the human contact—is responsible for the majority of these sporadic infections; such contacts may be persons incubating the disease, established carriers, or probably more commonly transient carriers who may not succumb to the infection but may excrete the organism for short periods. This possibility is well discussed in an excellent review of Salmonella infections by Savage (1956), who quotes one small investigation in which contacts in a number of sporadic cases of *Salm. typhi-murium* infection were found to be symptomless excretors of the organism.

Probably the majority of Salmonella infections occur as a result of the ingestion of unrefrigerated foods which have been infected by such human excretors, although no doubt other sources of infection may be incriminated in certain instances. The types of food eaten in the summer period are undoubtedly more favourable for the survival and multiplication of the organisms. There is good evidence to suggest that a person must ingest a considerable number of organisms before enteric symptoms will develop (Newell, Hobbs and Wallace, 1955); our own observations have tended to confirm this. The cold meats, meat pastes, salad creams *et cetera*, regularly consumed by many people during the summer provide admirable media for the rapid multiplication of the organism; even if such foods are left for periods of only a few hours at room temperature, an appreciable dose of Salmonellæ may have developed, perhaps even in only isolated sites of the foods. In winter hot meals are more generally eaten and less opportunity exists for the organisms to multiply in these types of food.

The incidence of salmonellosis probably could be restricted if food handlers (including the family cook) were instructed in better personal hygiene; the more general use of refrigeration would also greatly assist the reduction of incidents. Ideally, routine bacteriological examination of the contacts in each sporadic case of gastro-enteritis should be sought; but this could be accomplished only by close cooperation between the family doctor and the central laboratory. Such a measure would not only provide information of epidemiological value, but would also help to restrict the incidence of these infections in the general population.

Summary.

1. A record of the Salmonella species isolated from human sources in Victoria over the past six years is presented to indicate the general pattern of human salmonellosis in this State.

2. One thousand two hundred and fifty strains have been isolated in this period, of which almost 80% have been *Salm. typhi-murium*. Of next importance are *Salm. bovis-morbificans*, *Salm. adelaide* and *Salm. newport*. In all, 29 species of Salmonella have been isolated, two of which have been confirmed as new species.

3. The quarterly incidence is recorded, and certain features considered to be of epidemiological interest have been indicated.

References.

ATKINSON, N., and WOODROOFE, G. M. (1944), "The Occurrence of Salmonella Types in Australia: 1", *Australian J. Exper. Biol. & M. Sc.*, 22: 51.

ATKINSON, N., WOODROOFE, G. M., and MACBETH, A. M. (1947), "The Occurrence of Salmonella Types in Australia: 3", *Australian J. Exper. Biol. & M. Sc.*, 25: 25.

ATKINSON, N., WOODROOFE, G. M., and MACBETH, A. M. (1949), "The Occurrence of Salmonella Types in Australia: 4", *Australian J. Exper. Biol. & M. Sc.*, 27: 375.

ATKINSON, N., WOODROOFE, G. M., and CULVER, D. E. (1952), "The Occurrence of Salmonella Types in Australia: 7", *Australian J. Exper. Biol. & M. Sc.*, 30: 73.

ATKINSON, N., CARTER, M. C., WOLLASTON, J. C., and WALL, M. (1953), "The Occurrence of Salmonella Types in Australia: 10", *Australian J. Exper. Biol. & M. Sc.*, 31: 465.

COOPER, G. N. (1956), "A Rapid Method for the Identification of Salmonella Species", *J. Path. & Bact.*, 72: 39.

EDWARDS, P. R., and BRUNER, D. W. (1942), "Serological Identification of Salmonella Cultures", *Bull. Kentucky Agric. Exper. Station*, Circular No. 54.

MACKERRAS, M. J., and MACKERRAS, I. M. (1949), "Salmonella Infections in Queensland", *Australian J. Exper. Biol. & M. Sc.*, 28: 163.

MEDICAL RESEARCH COUNCIL OF THE PRIVY COUNCIL (1947), Special Report Series No. 260.

MILLER, A. A., NICOL, C. G., and RAMSDEN, F. (1955), "An Outbreak of Food Poisoning Due to Salmonella Bovis-morbificans", Medical Research Council of the Privy Council, Special Report Series No. 96.

MUSHIN, R. (1948), "An Outbreak of Gastro-Enteritis Due to Salmonella Derby", *J. Hyg.*, 46: 151.

NEWELL, K. W., HOBBS, B. C., and WALLACE, E. J. G. (1955), "Paratyphoid Fever Associated with Chinese Frozen Whole Egg", *Brit. M. J.*, 2: 1296.

SAVAGE, W. (1956), "Problems of Salmonella Food Poisoning", *Brit. M. J.*, 2: 317.

WILSON, M. M., and MACKENZIE, E. F. (1955), "Typhoid Fever Due to Consumption of Infected Desiccated Coconut", *J. Appl. Bact.*, 18: 510.

AN OUTBREAK OF INFECTIOUS HEPATITIS.

By J. G. WILSON,
Renmark, South Australia.

AT Renmark, on the River Murray in South Australia, an outbreak of infectious hepatitis occurred, beginning in April, 1955, and continuing until August, 1956. Seventy-two cases are here reviewed, and from them several interesting points emerge which I believe are worth recording.

Of the 72 patients, 39 were females and 33 males, with ages ranging from four to sixty years. It seemed an illness to affect the younger age groups, as 37% were aged twelve years or under.

The epidemic began in April, 1955, and slowly and steadily increased in magnitude, with the months of December, 1955, and January and February, 1956, as months of maximum incidence. After this the epidemic slowly subsided, only isolated cases occurring after June, 1956. It may be significant that a previous outbreak of infectious hepatitis in the district in 1950 also occurred during the summer heat. It is during these months that the river is often high and much stagnant water is about, and also the irrigation channels are full and in operation. This fact suggested a water spread of the epidemic.

In the symptomatology itself there was nothing of great significance. A distaste for tobacco in the pre-icteric phase was common enough to be worthy of mention. Subclinical cases were diagnosed and many others were suspected in view of the fact that the epidemic was present in the district. It was thought that the incubation period lay between two and four weeks. In three definite cases there was a delay of two to four weeks between members of the family contracting the illness.

In seven instances only out of the 72 cases did more than one member of the family contract the illness. This seems to confirm the method of spread of the disease as being via the gastro-intestinal tract, rather than by droplet infection from the naso-pharynx as is seen in poliomyelitis today.

The relapse rate was 4%, three patients only relapsing at about one month from the original illness. All three apparently have recovered completely in spite of being allowed up and about during the relapse. This figure is perhaps low in comparison with other reported figures of 20% or so. It may often be that the icteric illness in which one first sees the patient is really the relapse following a subclinical attack.

Of the series, one case was diagnosed as the anginose variety of glandular fever complicated by hepatitis. This patient was ill for three months, but eventually recovered. It is also of interest that many cases of glandular fever were seen in the area concurrently with this hepatitis epidemic. The close association between these two illnesses is known; perhaps the same or a variant of the same virus may be the ætiological agent.

The first patient seen was known to have a heterozygous twin sister in Sydney, who contracted the illness on the same day, also relapsed one month later, and definitely had no contact with her twin. The sister in Sydney was kept strictly in bed throughout the relapse, but recovered clinically no faster than my patient, who was ambulant throughout.

Several patients have continued to feel unwell for several months following the attack, but on the whole all have apparently recovered. It is difficult, of course, to establish any relation to a subsequent cirrhosis in these cases. None of the 1950 epidemic patients have since suffered from clinical cirrhosis. Also, Renmark being in the centre of the wine-growing area, the disease of cirrhosis of the liver is reasonably common; but to my knowledge no cirrhosis patients give a history of clinical hepatitis.

Interestingly enough, two of the 72 cases occurred in women in the last trimester of pregnancy—one a week and the other a day or so prior to confinement. Fortunately in both cases the mother and baby are well.

The cases occurred quickly in about December, 1955, with much stagnant water about, and the diagnosis of leptospirosis was suggested; but after several normal white blood cell counts had been found, and since in this epidemic there was no toxæmia or persistent pyrexia, jaundice was seen in all cases and there was an incubation period of approximately a month, this diagnosis was discounted.

The most interesting feature about this outbreak was the way in which the cases occurred at the start, and the manner in which the epidemic spread in and about Renmark. A large scale map of the area was obtained and the cases were plotted on it. It was evident that cases occurred as isolated events at first on the edge of the settlement, and soon it became evident that small groups of cases in almost isolated little communities occurred for the most part, outside the town itself in what is known as the settlement area. Of course, in these small groups a history of contact was frequent and of no definite value in epidemiology. As the epidemic progressed, people in the town itself contracted the illness, and it generally became fairly widespread. On studying the map of the plotted cases, on which is shown the irrigation channel system, it was suggested that the cases tended to some degree to occur along the major irrigation channels, and a method of spread along these lines was clearly possible.

Of course, it is well known that symptomless carriers are possibly the chief disseminators of the illness; but the isolation of the illness to little communities in the very early stages before it became too widespread—that is, in the first four months or so when personal contact was close—was an interesting feature. In these areas there is as yet no sewage system, and septic tanks are rare. This open-pan system of sewage disposal therefore may be a major factor in the spread of the illness. The water from the irrigation channels, although for the most part used for irrigating the land, is also used as drinking water to some extent, especially in midsummer, when rainwater tanks are low. From this it can be seen how it is clearly possible for a water-borne spread of the disease to occur, and the distribution of this epidemic did suggest a method along these lines.

Unfortunately it was impossible to follow up this study in any way, and I have nothing more than a clinical impression of the epidemiology of this interesting disease.

Reports of Cases.

A CASE OF CARCINOMA OF THE BREASTS WITH MULTIPLE METASTASES.

By HOWARD BULLOCK,
Sydney.

Let me not live after my flame lacks oil, to be the snuff of younger spirits whose apprehensive senses all but new things disdain.

NOTHING original is claimed in the treatment of this patient, but Mrs. X has had the advantage of the old surgical skill and of the new, the scientific use of X rays, and the administration of glandular extract.

In an address before the American College of Surgeons in October, 1928, the late Sir Charles Ballance, under whom I was privileged to work as a young man, paralleled tuberculosis with cancer. He pointed out that before 1881 tuberculosis was regarded as a disease due to many causes, as many authorities regarded cancer at the time of his address and continue to regard it. He considered that the analogy between tuberculosis and cancer was perfect. My generation considered cancer with disseminated metastases as unsuitable for operation.

Clinical Record.

In February, 1948, Mrs. X, aged thirty-eight years, consulted me for a tumour of the right breast which had been noticed a month previously. Examination of the patient revealed a hard tumour of the breast, but no glands could be palpated. The breast and glands were removed by radical operation a few days later. Pathological examination by Dr. A. Finckh revealed a schirrous cancer, but no involvement of the glands.

All went well for four years after operation, when a small sore appeared in the centre of the operation area; it gradually grew till the patient was examined by me in April, 1955, seven years after the operation. A fungating mass about four inches in diameter was present over the site of the operation. The patient looked very ill. She had been losing blood regularly for some months, and sepsis was present. A week before she consulted me, sciatic pain in the right leg had been felt; this suggested sciatic nerve involvement. X-ray examination of the skeleton was advised, but was refused.

The mass was excised by diathermy, and skin from the abdomen was switched to cover the raw area. The raw abdominal area was skin-grafted.

The operation was undertaken chiefly as a palliative measure, as the outlook seemed grave.

After much persuasion the patient submitted to X-ray examination of the skeleton by Dr. J. G. Edwards and Dr.

H. M. Cutler. Their findings were as follows: (i) consolidation and atelectasis at the base of the right lung; (ii) absorption of the bodies of the seventh cervical vertebra, and of the fourth and fifth dorsal vertebræ; (iii) some doubtful areas in the skull vault, and deposits in the right pelvic bone near the acetabulum and in the upper third of the right femur. Their conclusion was that "widespread carcinomatosis is present". Removal of the ovaries and of the suprarenal glands was recommended, but was refused. Bi-weekly injections of "Testoviron" have been given since.

The patient was referred to Dr. E. J. B. Hardcastle and partners for X-ray therapy; their report is as follows:

> Four ports were given from June 17 1955 to 18th July 1955 to the ovaries and pelvis.
>
> In July 1956, recurrent skin nodules on the left side of the chest were treated by deep X-ray from July 30th to August 3rd 1956.
>
> X-ray on 25th July 1956 revealed:
>
> Marked sclerosis of the body cervical vertebra seven, marginal lipping of dorsal vertebræ four and five. Chest, no change, lumbar spine, no change.
>
> Lesion in the neck of the right femur is completely sclerosed. Lesions on the right side of the pelvis show regeneration of the bone.
>
> No new lesions have appeared.

In July, 1956, a hard mass appeared in the left breast, but as the examination of the overlying skin revealed the presence of cancer nodules, operation was withheld.

As the skin after the X-ray therapy in July seemed to have recovered completely, the left breast and glands were radically removed in November. Dr. Eva Shipton reported the presence of schirrous cancer, but of seven glands examined, only one was found to be involved by cancer. This gland during operation was remarked on, as it was firmly encapsulated in fibrous tissue and to the experienced eye was cancerous.

The patient now looks remarkably well over two years after operation. She has gained three stone in weight. Her colour is excellent and her general health is good. The skin on the left breast area is pliable and appears normal. To all intents and purposes, with improvers on, the patient appears a person enjoying the best of mental and physical health. The bone metastases in this case have subsided, it is hoped permanently. Surgery has played no part in this case, apart from the removal of the main growths.

Comment.

> No human being is constituted to know the truth, the whole truth and nothing but the truth. And even the best of men must be content with fragments, with partial glimpses, never the full fruition.

Is the dawn breaking, giving a glimpse of the control, if not the cause, of cancer?

One thing is certain—that in the future surgery will play a minor role in the treatment of cancer.

Reference.

BALLANCE, C. (1928), "Some Thoughts on the Nature of Cancer", *Surg., Gynec. & Obst.*, 48: 297.

Reviews.

Clinical Hematology. By Maxwell N. Wintrobe, M.D., Ph.D.; Fourth Edition; 1956. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9¼" × 6", pp. 1186, with 236 illustrations and 20 plates, 18 in colour. Price: £8 5s.

A NEW EDITION of "Clinical Hematology" by Maxwell M. Wintrobe will be welcomed by clinicians as well as by hæmatologists. There is perhaps no field in medicine which has expanded more in recent years than hæmatology. Improved visual techniques such as microcinematography, combined with phase microscopy, have shown us cellular activities unobserved before. Study of the coagulation of the blood has revealed hitherto unimagined complexities, leading to the precise diagnosis of hæmorrhagic states and improvements in their treatment. The effect of ionizing radiation on the blood and bone marrow is of great importance, as are the effects of benzol and many chemical substances used in the industries and in the household life of our complicated civilization. These agencies are linked in some ways with the study of the leuchæmias. As Wintrobe observes in his preface, the preparation of a new edition of a book on hæmatology is becoming a more and more laborious task. In the fourth edition, as in previous ones, the literature has been most capably and comprehensively reviewed. The greater proportion of the text of the third edition has required modification, some of it quite extensively. Two chapters have been added, one on blood groups and blood transfusion, another on the abnormal hæmoglobin syndromes. The author states that the new material which has been added comprises approximately 800 typed manuscript pages. Several new colour plates have been added. Some of the tables of normal values have remained unaltered since the first edition in 1942. In view of the enormous amount of work that has been done since then, one might have expected some statistical modifications. The precise sources of these figures are not always stated. Some of Wintrobe's normal values differ from those given by British hæmatologists; for instance, the normal eosinophile cell count is given as 50 to 250 per cubic millimetre; Dacie's estimate is 40 to 440 per cubic millimetre, based on quoted surveys (possibly, racial and geographic conditions are responsible for a real variation). These are, after all, very minor matters; this book continues to be one of the two best text-books on hæmatology written in English—and in very good English, too.

Athletic Injuries: Prevention, Diagnosis and Treatment. By Augustus Thorndike, M.D.; Fourth Edition, thoroughly revised; 1956. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 8¼" × 5¼", pp. 252, with 113 illustrations. Price: 49s. 6d.

THIS book describes, in considerable detail, the author's experience as Surgeon-in-Charge of the Department of Athletics, Harvard University. It is divided into three parts, the first dealing with prevention of injuries, the second with general considerations of injuries, and the third with regional injuries, their diagnosis and treatment.

There is no doubt that, with careful supervision of training and early treatment of injuries, the time lost on account of athletic injuries can be greatly reduced, and the first part of the book will provide valuable advice to medical officers and physiotherapists who are interested in this sphere of work.

Sport in American universities is apparently of great importance: "Every day saved in convalescence is gold, each week saved is worth a three-carat diamond." The value in dollars is probably considerable also.

Extensive strapping procedures are amply described and illustrated for chronic sprains and strains, to protect the injury and allow an early resumption of sport. However, there will be many surgeons in this country who would rather allow a longer period of rest in order to obtain a natural repair of injured tissues so that recurrent sprains can be avoided. Many also will be disappointed to read that "heat and prolonged rest is the only logical treatment" for tennis elbow, and surprised that "anterior tibio-fibular ligament" sprains are the most common of ankle sprains.

This book will have a limited appeal to those practitioners entrusted with the care of the athlete, but may prove rather disappointing to those seeking advice on the details of treatment.

Handbook of Biological Data. Edited by William S. Spector; October, 1956. WADC Technical Report 56-273, ASTIA Document No. AD110501. Prepared under the direction of the Committee on the Handbook of Biological Data, Division of Biology and Agriculture, The National Academy of Sciences, The National Research Council. Wright Air Development Center, Air Research and Development Command, United States Air Force, Wright-Paterson Air Force Base, Ohio. 10¾" × 8¼", pp. 620.

SOME seven years ago the National Research Council of the United States National Academy of Sciences requested the Wright Air Development Centre to compile a handbook of established data, tables and fundamental measurements and attributes in biological science. Some 17,000 people, whose work could in any way be termed biological, and who could be regarded as expert in any particular field, have contributed their opinions on the most accurate information to be given. This information was shuffled and processed by

the editors, and the result is the handbook: 584 demy 4to pages. The information is vast in amount, and it is valuable information; but the handbook is not comprehensive; it never could be. Within this limitation it is a valuable book.

Modern Treatment Yearbook 1957: A Yearbook of Diagnosis and Treatment for the General Practitioner. Edited by Sir Cecil Wakeley, Bt., K.B.E., C.B., LL.D., M.Ch., D.Sc., F.R.C.S., F.R.S.E., F.R.S.A., F.A.C.S., F.R.A.C.S.; 1957. London: Published for the Medical Press by Baillière, Tindall and Cox, Limited. 8½" x 5½", pp. 320, with 24 plates and 23 figures. Price: 25s.

THE "Modern Treatment Yearbook" continues with its series of articles upon advancing topics. It does not attempt to be comprehensive, but does supply contributions from many English centres—not a few being provincial ones. This is always desirable in this sort of book. The standard is common sense; and the widespread sale of this book in the past is witness to its value.

The Year Book of Obstetrics and Gynecology (1956-1957 Year Book Series). Edited by J. P. Greenhill, B.S., M.D., F.A.C.S., F.I.C.S. (Honorary); 1956. Chicago: The Year Book Publishers, Incorporated. 7½" x 5", pp. 592, with 82 illustrations. Price: $6.75.

THIS Year Book, under the editorship of J. P. Greenhill, continues to give a useful review of obstetrics and gynæcology, and the abstracts are well chosen. A considerable amount of space is devoted to physiological and anatomical principles. Altogether this book is of great value to doctors working in this specialty as a reference to current opinion.

The Visual Fields: A Textbook and Atlas of Clinical Perimetry. By David O. Harrington, A.B., M.D., F.A.C.S.; 1956. St. Louis: The C. V. Mosby Company. Melbourne: W Ramsay (Surgical), Limited. 10" x 7", pp. 327, with 234 illustrations and nine colour plates. Price: £8 16s.

LIKE most of the Mosby Company publications, this is a handsome volume. The contents are sound and complete and reflect the good judgement and wisdom of the author. For the student and the ophthalmologist there is no more attractive book for giving a good working knowledge of the visual fields, familiarity with methods of measuring them and facility in interpreting the results. Apart from a few new methods, such as flicker frequency tests, stroboscopic stimulation, binocular extinction tests and multiple pattern methods, and a few new instruments, including the marvellous Goldmann perimeter, there has been little basic change in the taking or interpreting of fields for a good many years.

This book falls short of at least one well-beloved standard work on the same subject. Nevertheless, it is very good, beautifully laid out, magnificently illustrated and full of balanced wisdom. It is up to date and can be trusted and respected. The nine coloured plates are in themselves a course in anatomy of visual pathways and interpretation of fields. At the price, it is expensive; but if clarity, ease of study and lucidity have value, here it is.

Anæsthetic Accidents: The Complications of General and Regional Anæsthesia. By V. Keating; 1956. London: Lloyd-Luke (Medical Books), Limited. 8¾" x 5½", pp. 269. Price: 25s.

IN its 261 pages V. Keating's book, "Anæsthetic Accidents", gives a brief yet comprehensive survey of the disastrous possibilities associated with modern anæsthetic practice, including both local and general methods. A better title might have been "Anæsthetic Hazards", for rather than describing specific accidents, the work deals mainly with the physiological, pharmacological and technical origins of the numerous adverse reactions that may occur during anæsthesia.

Opening with a formidable examination of the autonomic and circulatory factors that may lead to shock and cardiac arrest, the book next deals exhaustively with the disturbances of respiratory function and the pulmonary complications that anæsthesia may occasion. Interesting comments follow on the hazards of barbiturate anæsthesia, appended to which is some reference to neurological complications, mainly concerning brachial palsies. The complications of local, and especially spinal, anæsthesia are then extensively and alarmingly reviewed. Some attention is now directed to the hazards of endotracheal anæsthesia, as well as to the ill effects of bad posturing of the patient during operation. A very good section on the action and side effects of the relaxing agents then follows, after which inadequate and largely irrelevant chapters on infection, explosions, safety precautions and medico-legal considerations are included. An excessively brief index completes the work.

In this book the obvious demand for brevity has in many places impaired the clarity of the presentation, although generally this is commendably lucid. Numerous references are quoted in the text and summarized at the end of each chapter. There are a few crudities of style and grammar, and occasional inconsistencies like the reference to "inanition" during the discussion of bronchial drainage on page 104. This could be a typographical error, which no doubt applies to "Ammophyllin" on page 99 and to "council of perfection" on page 114. Otherwise the format is excellent, the type is large and clear, the subdivisions are well indicated and strictly relevant, and the implications of the work are incontestable. Even if in many places it does seem to exaggerate the dangers of anæsthesia, it certainly provides a salutary reminder of the fact that in the induction and conduct of this abnormal state no light approach is ever permissible.

Progress in Clinical Medicine. By various authors, edited by Raymond Daley, M.A., M.D. Cantab., F.R.C.P., and Henry Miller, M.D. Durh., F.R.C.P.; Third Edition; 1956. London: J. and A. Churchill, Limited. 9½" x 6¼", pp. 434, with 36 illustrations. Price: 40s.

THIS book has many virtues; unlike many of its fellows it does not attempt to recur every year like the winter. It is concerned with advances in medicine which are established; it is not didactic; and it still believes in clinical medicine. In general its authors have produced a book of lucid, valuable chapters completed by key references. Quite rightly it does not attempt to be comprehensive, but should be read by all physicians, budding or otherwise. It is a pity that the first two chapters of the book mar it, as they do not reach the high standard of subsequent chapters.

Hypnotism and Crime. By Heinz E. Hammerschlag, translated from the German, and with a foreword by Professor John Cohen; 1956. London: Rider and Company. 7½" x 5", pp. 148. Price: 13s. 6d.

IN "Hypnotism and Crime" Dr. H. E. Hammerschlag discusses some of the misuses of hypnotism. For the effective use of suggestion, the stimulus must be of sufficient intensity or frequency to by-pass the critical faculties and work directly on subconscious mechanisms. Under ordinary circumstances a hynotic subject refuses to do anything which is gravely antisocial or damaging to his own self-respect. On the other hand, Dr. Hammerschlag reports cases in which crimes were committed, apparently in response to hypnotic suggestion, but only after the subject had been under the hypnotist's influence for lengthy periods, in one instance for several years. It is thus difficult to determine how far the criminal acts were committed under hypnotic suggestion, and how far under the evil influence of the hypnotists with whom the women concerned had formed liaisons. One wonders at the *naïveté* of a husband who continued to pay out large bills to a bogus doctor after his wife had threatened his life on several occasions, and of the neurologists and psychiatrists whose investigations "showed that there was no question of her being a psychopath, a hysteric or morally unbalanced". Her supposed amnesia for her homicidal conduct after the police had been called in was dealt with in series of investigations under hypnosis by a qualified psychiatrist. Dr. Hammerschlag describes some of the ways in which stage hynotists select their "guinea-pigs", and gives a warning about the undesirable effects which may follow these mass demonstrations. He does not refer to the orgiastic responses to certain "crooners". There is a terrifying account of the extortion of a "confession" from an Austrian physicist after several days of continuous "brain bashing". This little book is of special interest to psychologists, criminologists and psychiatrists, and is also recommended for its entertainment.

Industrial Deafness: Hearing Testing and Noise Measurement. By Joseph Sataloff, M.D., D.Sc. (Med.); 1957. New York, Toronto, London: The Blakiston Division, McGraw-Hill Book Company, Incorporated. 9" x 6", pp. 347, with illustrations. Price: $8.00.

IN this book, subtitled "Hearing Testing and Noise Measurement", the author, assisted by specialist contributors in physical acoustics, covers in one volume all phases of industrial deafness. He states: "It is intentionally fundamental and is directed not to the acoustical expert but to

industrial executives, industrial physicians, hearing testers, otologists, nurses, industrial hygienists and engineers."

Because so many people with different training are involved, much of the information on this subject is spread through the specialized medical, physical, physiological and audiological literature, as well as in reports of specialized committees or organizations. This up-to-date volume adequately brings together the fundamental information required for the necessary combined attack on this problem.

Because of the wide individual differences in susceptibility to noise, industrial deafness must be attacked through hearing conservation rather than through noise measurement—the latter being used to decide where a hearing conservation programme is necessary or desirable. The emphasis is on this most important aspect of the need for and implementation of a hearing conservation programme; this is treated in detail.

Recent information on the allowance for presbycusis, on damage risk criteria and on medico-legal developments will be found most valuable.

This book is recommended to all concerned with industrial deafness.

A Practical Handbook of Psychiatry for Students and Nurses. By Louis Minski, M.D., F.R.C.P., D.P.M.; Third Edition; 1956. London: William Heinemann (Medical Books), Limited. 7¼" × 5", pp. 152. Price: 7s. 6d.

THIS book has apparently found acceptance amongst those for whom it was intended, as after only ten years it is now in its third edition. However, although the author has attempted to bring it up to date, he has not overcome the difficulty of packing into a short volume the essentials of psychiatry for both medical students and nurses. As a result it is inadequate for the one and probably too much for the other. Medical students may find it useful for revision purposes.

Illustrations of Bandaging and First-Aid. Compiled by Lois Oakes, S.R.N., D.N. (Leeds and London); Fifth Edition; 1956. Edinburgh and London: E. and S. Livingstone, Limited. 8½" × 5⅝", pp. 333, with 396 photographs. Price: 12s. 6d.

THIS book, which we reviewed favourably when it first appeared in 1940, is now in its fifth edition. Its main virtue is its comprehensive and well-illustrated section on bandaging.

Human Disease. By A. E. Clark-Kennedy; 1957. Victoria: Penguin Books Proprietary, Limited. 7" × 4¼", pp. 267. Price: 5s. 6d.

A. E. CLARK-KENNEDY is well known for his lucid and attractive writing on fundamental aspects of medicine. In this book he has turned from his usual medical readers to the general public in an attempt to explain the nature of human disease. His own formula is that he has "endeavoured to describe human disease in terms of what it really is, namely, reactions, some common others rare, many complicated and little understood, between the individual man and his environment". The result is as happy as one could reasonably wish for in what must have been an extremely difficult task. The intelligent non-medical reader should find the reading of this book well worth the effort required in dealing with medical terms and unfamiliar ideas. Most doctors will enjoy and profit from it, especially those parts dealing with basic principles.

Fundamentals of Clinical Fluoroscopy: With Essentials of Roentgen Interpretation. By Charles B. Storch, M.D.; Second Revised Edition; 1957. New York: Grune and Stratton, Incorporated. 10" × 6", pp. 320, with 318 illustrations. Price: $8.75.

THIS is the second edition of a text first published in 1951, and is written primarily for the "internist, pediatrician and general practitioner", who wish to conduct their own fluoroscopic examinations. As such, it is quite a practical manual full of useful details in the management of the fluoroscope and the diagnosis of lesions in the chest and abdomen. The training registrar in medicine, surgery or radiology will also find much of interest.

The first chapter deals briefly with the basic concepts—the mechanics of fluoroscopy, protection and dark adaptation. The remainder of the book consists of eight chapters, each dealing with a particular section of the body. There are chapters on the chest, heart, œsophagus and bowel, and there is also a section devoted entirely to the gall-bladder. Radiographic appearances and normal anatomy are discussed together; but we could wish that there had been better correlation of abnormal "shadows" and the underlying pathological condition. Reproductions are generally of good quality, and, in the main, are typical of the fluoroscopic images of various disorders and conditions. There has been rather an excessive amount of retouching, which might have been eliminated with more careful selection of the original radiographs.

The book will have little appeal to radiologists, many of whom will consider that it gives a false idea of the importance of this aspect of radiological investigation, and may possibly lead to much unnecessary fluoroscopy. As the author states, "of the various diagnostic X-ray applications, fluoroscopy probably represents the greatest radiation hazard, not only to the examiner but to the patient". Whether, in view of the present-day attitude to radiation dangers and genetic effects, the practice is one to be encouraged, particularly in less experienced hands, is open to question. In any case, "as no diagnosis should ever be made by fluoroscopy alone", it would appear that in many instances it would be better to proceed straight to the taking of the radiograph.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Studies on the Distribution of Tritium-Labelled Dihydrostreptomycin and Tetracycline in the Body", by Torsten André; 1956. Stockholm: Acta Radiologica (Supplement 142). 9¾" × 7", pp. 92, with 41 illustrations. Price: Sw. Kr. 25.

The account of a Swedish investigation.

"Expert Committee on Addiction-Producing Drugs: Seventh Report." World Health Organization Technical Report Series Number 116; 1957. Geneva: World Health Organization. 9¾" × 6¾", pp. 16.

An account of a meeting held in October, 1956.

"The Technique of Fluid Balance: Principles and Management of Water and Electrolyte Therapy", by Geoffrey Tovey, M.D.; 1957. Edinburgh and London: Oliver and Boyd. 8¾" × 5¾", pp. 108, with tables.

A short account for the busy doctor, especially the one with no laboratory facilities.

"Group Psychotherapy: The Psycho-Analytic Approach", by S. H. Foulkes and E. J. Anthony; 1957. London: Penguin Books, Wyman and Sons, Limited. 7" × 4¼", pp. 277. Price: 5s. 6d.

Written for the lay reader.

"The Surgical Clinics of North America: Chicago Number: February, 1957: Modern Orthopedic Management", edited by Edward L. Compere, M.D.; 1957. Philadelphia and London: W. B. Saunders Company. 9" × 5¾", pp. 302, with 168 illustrations. Price: Cloth binding, £8 2s. 6d. *per annum*; paper binding, £6 15s. *per annum*.

Contains 19 contributions relating to modern orthopædic management.

"Eleven Lourdes Miracles", by D. J. West, M.B., Ch.M., D.P.M.; 1957. London: Gerald Dickworth and Company, Limited. 8¾" × 5⅝", pp. 144. Price: 15s.

"An examination of the medical documents relating to eleven 'miraculous' cures at Lourdes, 1937-1950."

"Premier Symposium de la Fondation Valentino Baldacci (Madrid, 27-30 Mai 1955); Hémostase spontanée, plaquettes sanguines et paroie vasculaires", published by J. Roskam; 1956. Pisa: Omnia Medica Éditeur. 9¾" × 6¾", pp. 192, with five illustrations.

The account of a hæmatological symposium with notable contributors from England and the Continent.

"Roentgen Changes in the Cranium in 153 Intracranial Tumours in Children aged 0-15 Years", by Helge Hertz, M.D., and Thomas Rosendal, M.D.; 1956. Stockholm: Acta Radiologica (Supplement 141). 9¾" × 7", pp. 56, with 32 illustrations. Price: Sw. Kr. 30.

The title is self-explanatory.

The Medical Journal of Australia

SATURDAY, JUNE 15, 1957.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE WELFARE OF ABORIGINES IN WESTERN AUSTRALIA.

MUCH publicity has been given recently to a report by a Select Parliamentary Committee on the health and nutrition of the natives in the Warburton Range area of Western Australia, which the committee visited. This committee was not specially qualified to make observations on disease or nutrition, and it was desirable that further information on the subject would be forthcoming from those trained to assess these matters. For that reason it is reassuring to learn that the findings of the members of a subsequent medical expedition to the Warburton area were not so gloomy as those of the Select Parliamentary Committee. The full report of the medical party is not available; but in view of the widespread interest in the matter and particularly of the misconceptions that have been aroused, some reference should be made at this stage to a preliminary report of the medical party which has been prepared at the request of the Minister for Native Welfare in Western Australia. The party consisted of five members: Dr. W. S. Davidson, Deputy Commissioner of Public Health for Western Australia; Professor Ida Mann, Consultant Ophthalmologist to the Department of Public Health; Mrs. Mezger, Secretary to Professor Mann; Mr. R. Davis, Chief Technician of the Department of Hæmatology in the Royal Perth Hospital; Miss W. Moyle, Health Inspector in the Department of Public Health.

The two main aspects of the investigation were a follow-up study of a previous trachoma survey made by Professor Mann in 1954, and an evaluation of the nutritional state of the natives and of the prevalence of disease amongst them. The eye examination, which was carried out by Professor Mann, was either full (277 natives) or partial (206 natives). The full examination included the search for infection and for signs of vitamin or nutritional deficiency, the determination of visual acuity, tests for colour blindness and an examination of the fundus in doubtful cases. In the partial examination, tests for visual acuity and colour blindness were omitted. The clinical examination was directed to the detection of infectious diseases and nutritional deficiencies; 300 natives underwent this examination. Samples of blood were taken from 160 natives and examined for hæmoglobin value, serum protein content, indication of nutritional or other anæmias, vitamin B_{12} and folic acid content and serum reactions indicative of yaws or syphilis. In the report Dr. Davidson stresses the importance of eye examinations in determining the presence of nutritional disease; and we must agree with him that with the rigorous clinical blood and eye examinations carried out it is impossible that evidence of starvation could escape notice. Dr. Davidson asserts firmly that no manifestation of starvation or any condition remotely resembling starvation was elicited from the natives presented for the examination. He goes on to state that this does not mean that dietary deficiencies were not detected. A few cases of vitamin and protein deficiency of a mild nature were seen, but they were entirely confined to natives living in association with civilization and were due to dietary inadequacies that had arisen from a reliance on some items of food obtained from missions or from the township of Laverton as a substitute for items in their natural diet. There is also a strong possibility that isolated groups of natives in the course of their wanderings encounter periods of food and water shortage and some hardship, but there is no condition of starvation that can be applied as a generalization to the natives in the Warburton area.

The findings in the examinations are set out briefly but in sufficient detail to substantiate these statements. As a full report will be available later, we need refer only to a few particular points at this stage. One important point made by Dr. Davidson is in relation to the enlargement of the abdomen as a sign of malnutrition or starvation. It is certainly true that an enlarged abdomen is a sign of dietary deficiency in some conditions, for example, kwashiorkor or beriberi, but neither of these conditions was detected in the natives examined. An enlargement of the abdomen was found to be almost universal and occurred in large male natives weighing 12 to 15 stone as regularly as in small children. The explanation offered by Dr. Davidson is that this enlargement is in part postural and partly a natural characteristic, probably an adaptation of a hunting community accustomed to having large meals at infrequent intervals, in contradistinction to the civilized habit of having frequent meals of moderate size. Another fact that has tended to be misinterpreted is the prolonged suckling of infants by their mothers until the infants are two or three years old. This has been cited as an indication of a lack of food in the area, but Dr. Davidson points out that it is just the opposite, as no woman can continue to lactate when receiving an inadequate diet. Rather is it evidence simply of the lack of a substitute for mother's milk for the child who is

being weaned and yet is not able to cope with the food of the older members of the tribe. It seems clear that any evidence of food deficiency is the result not of lack of food, but rather of the availability of sugar and flour on stations and missions; this reduces the incentive for the natives to gather their own native food, which is often more satisfactory.

Examinations of the natives for infection revealed no cases of leprosy or syphilis. Evidence of yaws was found in 25% of the blood samples examined; but the disease was of a mild character, and no disabling manifestations of tertiary disease were seen. A few children with acute middle ear disease were being treated at mission hospitals. The presence of head lice was less than among natives and half-caste natives in country towns. Trachoma was the only eye disease of importance, but secondary infection was almost entirely absent, so that the disease was relatively mild. Secondary infection, which is responsible for much of the damage to eyes associated with trachoma, would appear to be the result of contact with white civilization.

Dr. Davidson points out that the country between Warburton and Blackstone is mulga-spinifex country with numerous hill ranges; "by no stretch of the imagination can it be termed desert". Game, in the form of rabbits and kangaroos, seemed plentiful, there was no lack of mulga seeds and quandongs, and there were probably numerous other seeds and berries known to the natives. All natives examined had had contact with the missions or other outposts of civilization, and in this regard it is interesting to note Dr. Davidson's acknowledgement of the valuable work done by the matrons of mission hospitals and of the important part they play in the health of the natives. He states that during the investigation no real bush native was seen and considers it very doubtful if such natives exist. It is important to note that the native camps were "indescribably filthy, every rudiment of sanitary science being ignored".

The conclusion of the report is that the natives in the bush have a more balanced diet and suffer from less infection than those in close contact with white civilization. Unfortunately, the natives seem irresistibly attracted to centres of white man's culture and tend to end up as unemployables in squalid natives' camps. Dr. Davidson attributes this not so much to any fault in the educational arrangements or the missions, as to a lack of agreement on the final function of the aboriginal in Australian society and the means of attaining this function. He regards it as beyond his jurisdiction to comment on native policy, but this is so intimately bound up with the health and welfare of the aborigines that his comments should be heeded. It does seem, as he remarks, that there is no turning back and returning of the native to his isolation and primitive life in native reserves. Whatever course is adopted in educating the native to play his role in Australian society, the lessons of this survey must be borne in mind. His education must include such things as diet, hygiene and sanitation, and a sense of responsibility for his own health, his family's health and the health of the community. Otherwise, in his new environment, he will become a prey to disease and a danger to others. How his affairs are to be arranged is essentially a matter for the administrative authorities, and it would be unfair to suggest that this is not receiving serious and responsible thought at both the Federal and State levels, whatever may have been our deficiencies in this regard in the past. It is sincerely to be hoped that a sound, coordinated, Australia-wide policy will be evolved by those best fitted to determine it, and that the power will be given to put it into effect.

Current Comment.

BACK PAIN AND X-RAY SPONDYLOSIS.

IT is a common perplexing task to sort out how much of back pain is due to the changes that X-ray examination has detected. In the presence of detected changes the clinician is still left wondering, because many patients with severe radiological spondylosis do not complain of pain. Frequently, however, he is glad to have something to blame and becomes convinced that his search has successfully concluded. Does pain occur with spondylosis? Or with degenerative spinal joint disease? If so, how frequently does it occur? Spondylosis is denoted by X-ray changes of bony spurs of the vertebral bodies, narrowed disk spaces and narrowed intervertebral foramina, occurring singly or in various combinations. The only true joints of the spine are the apophyseal and costo-vertebral, and from a purist point of view these are the only joints capable of showing hypertrophic and degenerative changes of the type elsewhere labelled osteoarthritis.

The subjects of the few and controversial reports in which investigation has been undertaken have been either persons seeking employment or suffering from pain, or patients with various illnesses. None of these satisfy objective criteria for answering the questions posed. A. E. Hussar and E. J. Guller[1] have attempted to sample a population free from motivation in respect of their backs. All those with compensation claims, or with a history of significant back injury, rheumatoid arthritis or other systemic disease, were rejected. Any who on physical or X-ray examination were found to have significant abnormalities other than spondylosis were rejected. The group consisted of 500 employees from all ranks in the Veterans Administration Hospital, Montrose, New York. They ranged in age from eighteen to sixty-five years, with an average of thirty-six years. The sex incidence is not stated. Clinical and X-ray examinations were conducted by each author separately, and neither author knew his colleague's findings until all data were collected.

Each subject was asked about the amount of physical work done, injuries sustained and pain experienced in the cervical or lumbar regions and adjacent areas, including pain radiating into the extremities. Physical examination was made for excess weight, limitations of movement, lumbar and cervical tenderness, and anomalies which might render the subject ineligible for study. X-ray examination included preparation of films of the cervical and lumbar areas in antero-posterior, lateral and two oblique positions. Films were examined for the presence of bony spurs, narrowing of intervertebral foramina and abnormalities of the apophyseal joints. Any other deviations from normal eliminated the subject from the study. Independent statistical analysis of the results was carried out.

Eighty-four subjects had spondylotic changes in the cervical part of the spine, in 13 cases associated with pain; 25 had pain without such changes. The correlation between pain and X-ray changes was of such a low order as to make it useless in prediction in an individual case. Correlation of severity of X-ray findings (spurs only; spurs and narrowed disk space; spurs, narrowed disk space and narrowed foramina) and incidence and severity

[1] *Am. J. M. Sc.*, November, 1956.

of pain (infrequent localized, frequent localized, frequent localized and radicular) was statistically not possible because of the low pain incidence. However, no subjects had radicular pain, and only three had frequent localized pain among the 63 who had narrowed disk spaces. Of this latter group 31 had narrowed foramina as well, and 23 had intraforaminal bone spurs. Age correlated highly significantly with positive X-ray findings, and just better than chance with pain. There was no correlation between overweight and X-ray findings or pain. There was a better than chance correlation between heavy work and X-ray findings, but not with complaint of pain. Hypertrophic changes in facet joints were seen in only two subjects, who were not complaining of pain.

With regard to the lumbar part of the spine, there was no correlation between X-ray findings and pain. Ninety-five subjects complained of pain, and 115 had X-ray signs, but only 26 of the latter had pain. It should be noted that no X-ray evidence of narrowed intervertebral foramina or intraforaminal spurs was found in this group; so that the radiological severity was assessed only in terms of spurs, early or advanced, and spurs *plus* narrowed disk space. Pain grading was as for cervical pain. Again statistical analysis was not possible because of the small numbers with pain. Only 13 had radicular pain, 10 of these without any X-ray evidence; the 17 with X-ray evidence of narrowed disk space had no radicular pain. Age, weight and heavy work correlated highly significantly with X-ray change, but not with pain. Hypertrophic facet-joint changes were seen in four subjects, along with spondylotic changes; two complained of pain, two did not.

In this series it was obvious that X-ray findings had no significance as far as providing a reasonable explanation for pain was concerned. Especially interesting were the 23 cases of intraforaminal cervical spurs with no root symptoms or signs, other than local neck pain in a few cases. The low incidence of true osteoarthritis was also noteworthy. Doubtless the spate of back X-ray examinations will continue on the basis of the many other possible bony abnormalities that infrequently give rise to back pain. This study in many ways serves as a base sample for other series orientated on specific disabilities, and has clearly shown the notorious futility of X-ray examination as an aid in investigating the commoner affections of the axial musculo-skeletal structures.

PULMONARY COMPLICATIONS AND PERFORATED PEPTIC ULCERS.

PULMONARY COMPLICATIONS, and in particular pulmonary lobular collapse, are well known as post-operative phenomena, and have usually been regarded as most likely to occur as a consequence of operative interference in the superior parts of the abdominal cavity. B. T. le Roux[1] noticed during a survey of these complications that the incidence amongst patients admitted to hospital with perforated peptic ulcer was extremely high. As a result of this observation, the pulmonary complications of perforated peptic ulcer were studied. Over a period of six months, fourteen patients underwent operation for perforation. Although these patients had radiographs of the diaphragmatic region taken, no radiological examination of the lungs was made. Of the fourteen patients, seven had post-operative pulmonary complications; and in each case the complication was pulmonary collapse. The complications were recognized clinically and confirmed radiologically.

In a subsequent period of six months sixteen patients were admitted to hospital with perforation of a peptic ulcer, and of these fourteen underwent operation. The latter received an X-ray examination of the chest on admission, whilst at the end of the operation bronchoscopy was performed and the amount of bronchial secretion obtained was measured. Of the radiographs, nine showed lobular collapse and two lobar collapse. At bronchoscopy more than one millilitre of mucopus was collected in the trap attached to the sucker.

Perforation of a peptic ulcer, which is almost always extremely painful, is known to cause considerable respiratory embarrassment, and this predisposes to pulmonary collapse. Pulmonary disease and smoking, both of which are common in people who suffer from perforated ulcers, complete the predisposing causes. Le Roux contends therefore that pulmonary changes complicate perforation usually before operation. This contention is borne out by the high incidence of pulmonary complications associated with conservatively treated ulcer perforations (A. D. Beattie; W. G. Gill and T. P. N. Jenkins).[1,2] The practical consideration brought forward by le Roux is for bronchoscopy and aspiration at the end of the operation for repair of the perforation.

[1] *Brit. J. Surg.*, January, 1957.

MUCORMYCOSIS.

MUCORMYCOSIS is a disease caused by certain fungi, notably *Rhizopus*. The fungi are common contaminants and are not normally pathogenic. Not a great deal is known about this disorder as it is by no means common. However, it appears to be increasing in frequency. Predisposing causes appear to be uncontrolled diabetes, leuchæmia, antileuchæmic drugs, some antibiotics and cortisone. The list perhaps is not surprising, nor perhaps is it surprising to watch the evolution of a new pattern of disease. The disease has been reported from many parts of the world, and is probably world wide. R. D. Baker[3] has recently discussed the disease and urged awareness of it. Involvement of the upper part of the respiratory tract is probably the commonest form of occurrence, but ocular, pulmonary, cerebral, intestinal and disseminated forms have been reported. Diagnosis depends upon awareness and demonstration of the fungus.

TO WHOM IT MAY CONCERN.

NAGGING is never popular amongst those who are at the receiving end, but without persistent reminders such productions as a medical directory would never appear. The 1957 edition of "Medical Directory of Australia" is about to be set in type, and it is hoped that the volume will be completed before the end of the year. Cards have been sent to all those whose names appeared in the last edition, and also to those whose names, for one reason or another, have appeared on the various State medical registers in the succeeding period. The response has been by no means complete. No doubt many doctors are quite satisfied with the entry as it stands and have not thought it necessary to reply for that reason. Others have, perhaps, procrastinated. Experience in the past makes it fairly safe to prophesy that after the new Directory appears, some members of the profession will declare that they have never received a card. However, the office of the Directory has done its best to make contact with all who might reasonably expect to have their names included in the Directory, and a last-minute appeal is now made to all doctors to return the cards that have been sent to them. Any who have lost their cards or who think that they have been overlooked are invited to get in touch with the office of the Directory at The Printing House, Seamer and Arundel Streets, Glebe, New South Wales.

Members of the medical profession are warned that the onus of the correctness of an entry in the Directory is on the individual and not on the publishers.

[1] *Brit. M. J.*, February 25, 1950.
[2] *Brit. M. J.*, May 5, 1951.
[3] *J.A.M.A.*, March 9, 1957.

Abstracts from Medical Literature.

THERAPEUTICS.

Sulphonamides.

W. P. BOGER, C. S. STRICKLAND AND J. M. GYLFE (*Antibiotic Med. & Clin. Ther.*, November, 1956) discuss a new sulphonamide, sulphamethoxypyridazine or "Kynex". This drug is well absorbed from the gastro-intestinal tract, and slowly excreted. Patients were given 1·0 to 4·0 grammes of "Kynex" by mouth, and the blood and cerebro-spinal fluid concentrations were noted. Satisfactory concentrations in the blood were observed after one dose of 2·0 grammes and the drug was still present in the blood seven days afterwards. Cerebro-spinal fluid concentrations were satisfactory in normal patients after a dose of 2·0 grammes. The acetylated form of the drug was rather insoluble, and its appearance in quantity in the urine was observed. This indicated the need of care in its administration. At present the drug is only in the experimental stages.

Penicillin Reactions.

R. W. BABIONE (*Antibiotic Med. & Clin. Ther.*, November, 1956) discusses the penicillin reaction rate in the United States Navy. Reactions to injectable penicillin declined from 2114 in 1951 to 861 in 1955. There were two deaths by anaphylaxis after intramuscular penicillin therapy in 1955. Of all reactions to antibiotics, 97% were due to penicillin. Reactions to injected penicillin were much more numerous than to oral penicillin, which has been used much more since 1952. The author appears to think that the decline in reaction rates in the United States Navy has been largely due to the fact that much less penicillin has been used in recent years.

Promazine Death.

J. J. WOODWARD AND J. D. SOLOMON (*J.A.M.A.*, December 1, 1956) report a case of fatal agranulocytosis during promazine therapy. Promazine is chlorpromazine without the chlorine atom. The dose was 0·10 to 1·0 gramme daily for a patient with a schizophrenic reaction, paranoid type. After seven weeks' therapy fever and *petit mal* were noted. The white blood cell count fell to 600 per cubic millimetre, with no mature neutrophile cells. Sternal marrow puncture showed arrest of the granulocytic series, only myeloblasts being seen. Tetracycline, ACTH and cortisone were given. There was no response, and the patient died of agranulocytosis on the forty-eighth day of treatment with promazine.

Pentaerythritol Tetranitrate.

T. M. FEINBLATT AND E. A. FERGUSON (*New England J. Med.*, February 21, 1957) describe a study of the absorption and clinical effects of pentaerythritol tetranitrate. Capsules called time-disintegration capsules, containing 30 milligrammes of pentaerythritol tetranitrate, were given every twelve hours. Barium sulphate was given in capsule form at the same time, and radiographs were taken at intervals to demonstrate the position reached in the bowel at those times. Blood level studies showed that there was no rise in nitrate level during the first hour, but after one and a half hours there was a definite rise, and relief of cardiac pain was noted. The capsules were given to patients with anginal symptoms on effort or at rest. The capsules are said to contain a water-soluble non-metabolite with four portions each of 7·5 milligrammes of pentaerythritol tetranitrate.

NEUROLOGY AND PSYCHIATRY.

Cervical Myelopathy.

E. CLARKE AND P. K. ROBINSON (*Brain*, September, 1956), writing on cervical myelopathy as a complication of cervical spondylosis, exclude those cases in which the myelopathy followed acute protrusion of the disk. They state that the role of acute trauma (in chronic cases) is varied. It may produce: (i) the acute protrusion syndrome, (ii) secondary spondylosis, (iii) cord compression in a spondylotic but symptomless spine; established myelopathy may be aggravated by trauma. The reason why only certain individuals with cervical spondylosis develop myelopathy is obscure, but the blood supply of the cord, aging of the cord, cord ischæmia due to associated cardio-vascular disease, the relative size of the spinal canal and the presence of congenital bony anomalies must all be considered. In reviewing 120 cases the authors found the following presenting symptoms: (i) motor symptoms in one leg, (ii) motor symptoms in both legs, (iii) sensory symptoms in the arms or hands, (iv) pain in the neck, shoulders or arms, (v) electric shock symptoms, (vi) sensory symptoms in the legs, (vii) motor symptoms in the arms. The differential diagnosis was from disseminated sclerosis, cervical cord neoplasm and motor neuron disease. Myelography is the most important single investigation. Treatment consisted in either medical treatment with a well fitting plastic collar, or if this failed, surgical treatment—laminectomy with section of the dentate ligament.

Clinical Features and Pathology of Post-Concussional Vertigo.

M. S. HARRISON (*Brain*, September, 1956), when discussing clinical features and pathology of post-concussional vertigo, states that giddiness is a prominent feature of the post-concussional syndrome, of which headache, emotional lability and difficulty in concentration are the other symptoms. In the majority, the giddiness is ill defined. In a minority of cases there is evidence of a labyrinthine injury with X-ray evidence of a labyrinthine fracture accompanied by severe deafness and loss of caloric responses. The authors reviewed 108 patients admitted to hospital with concussion following a head injury without serious skull fracture. In 91 of these the dizziness was slight, and recovery occurred in two to three months. In 17 cases positional nystagmus of the benign paroxysmal type was present, and in these a rotational type of dizziness was complained of; the recovery period was about one year. The medico-legal implication of these findings would appear to be considerable, as positional nystagmus may be a serious cause of incapacity for work after a head injury.

Mental Deficiency.

D. VAIL (*Am. J. Psychiat.*, August, 1956) discusses the problem of mental deficiency generally, and as observed in an institution. He observes that the personality of the mental defective is superficially similar to that of persons of normal intelligence. He is in contact with reality and experiences the same feelings of fear, hope, rage and affection. His affects are more crudely organized, less elaborate and less subject to normal modulation and control. His system of defences is faulty, he is prone to act out his impulses without control, or is subject to rigid and brittle repressions, which produce severe inhibitions in everyday functioning. Depression is the most urgent affective problem. He is sensitive and quick to detect hostility and react to it, and frequently he feels inadequate, sad and lost. Verbal communication is poor, and one is forced back to the use of less sophisticated therapeutic tools. The author advocates a *milieu* therapy, a relatively stable and compact group of workers implementing a comprehensive rehabilitation programme. This includes occupational, recreational and vocational training, and group and individual psychotherapy, thereby bringing to the patients gratifications of stable family life and normal social pleasures. An adjustment to the reality demands is stressed—namely, a permanent dependence upon society and an acceptance of this role compatible with self-respect and a useful life. By this means it is considered that the attitude, behaviour and performance of the mental defective can be modified.

Pearly Tumours in Relation to the Central Nervous System.

J. S. TYTUS AND J. PENNYBACKER (*J. Neurol.*, November, 1956) discuss a series of 41 pearly tumours in relation to the central nervous system. They state that these tumours arise from epithelial rests that result from defective closure of the neural tube. They may occur in the mid-line, laterally, or in epidural, intradiploic or pericranial sites. They are benign encapsulated tumours that usually do not invade surrounding regions. The most frequent sites of occurrence are in the subarachnoid cisterns at the base of the brain, also in the pericranial, orbital, petrous and diploic regions and in the cerebello-pontine angle. They are also seen in the cerebral cortex, cerebellum and spinal canal. The X-ray picture usually shows a circumscribed area of bony erosion from without inwards, with smooth, thickened bony margins. In the orbit, proptosis is the main complaint, and its unsightliness is the indicator for operation. With tumours in the petrous bone the patients usually present with progressive peripheral facial paralysis and associated

deafness and absent caloric reflexes on that side. In the intracerebral cases epilepsy is often a presenting complaint and may exist as the only symptom for many years. The cerebro-spinal fluid protein is usually normal. As the tumours are related to the subarachnoid or lateral ventricles, the ventriculogram shows the usual signs of a space-occupying mass with scattered collections of air dispersed throughout the tumour, mainly on its periphery and giving the appearance of a cauliflower. In the post-operative period there is often pleocytosis in the cerebro-spinal fluid or aseptic meningitis. The tumours can recur, as, while total removal of the majority of extradural tumours is possible, intradural tumours are densely adherent and may be adherent to or may invade vital structures.

An Assessment of Chlorpromazine and Reserpine.

H. FREEMAN (*New England J. Med.*, November 8, 1956) criticizes treatment with the new tranquillizing drugs chlorpromazine and reserpine. In his series these drugs were used in patients with aggressive, hostile or antisocial trends. The daily doses of reserpine were from 2·0 to 5·0 and even 10·0 milligrammes for four to six weeks. Both drugs had a tranquillizing effect superior to placebos. Anxiety states and chronic schizophrenia were treated, to the number of 700 or 800 patients. Reserpine and chlorpromazine seemed to be equally effective. The author states that there were very few striking improvements in the drug-treated group, yet he definitely supports these treatments; side effects of the drugs are regarded as unimportant apart from agranulocytosis. Sensitivity, constipation, hyperthermia, dermatitis, confusion, convulsions and jaundice are said to be unimportant effects of chlorpromazine, and drowsiness, nasal congestion, diarrhœa, bradycardia, hypotension, œdema, confusion, convulsions and the Parkinson syndrome are said to be insignificant effects of reserpine. The author states that all these side effects disappear with time or when appropriate treatment is given.

The Psychopath in a Mental Institution.

S. K. EHRLICH AND R. P. KEOGH (*Arch. Neurol. & Psychiat.*, September, 1956) discuss the answers to two questions: (i) What are the characteristics of those psychopaths who find their way to a mental institution? (ii) What is the clinical difference between the psychopaths with a normal brain-wave pattern and those who have an abnormal electroencephalogram? They studied a group of 50 psychopaths who were admitted to a mental institution, by a correlation of clinical, biographical, symptomatological, electroencephalographic and psychometric factors. The psychopaths with cerebral dysrhythmia (80%) come for treatment frequently of their own accord. They show signs of physiological immaturity and homœostatic disturbance in their brain-wave pattern. The psychopaths with a normal electroencephalogram (20%) appear to be more deviant, but also more stabilized, in their anomalous personality pattern. They come for treatment unwillingly and late. Their measurable attitudes show a very high degree of social maladjustment, and the prognosis for them is poor.

N-methyl - a, a - methylphenyl - succinimide in Psychomotor Epilepsy.

F. T. ZIMMERMAN (*Arch. Neurol. & Psychiat.*, July, 1956) reports the use of N - methyl - a, a - methylphenylsuccinimide on psychomotor epilepsy. There were 35 cases in his series. The average dose was four 0·3 gramme capsules per day—the range being from one to eight capsules per day. Toxic signs appeared in five patients. These were fatigue, loss of appetite, dizziness and periorbital œdema. No blood dyscrasias were observed over a three-year period of study. Seventy-four *per centum* of the patients treated showed a reduction in psychomotor seizures, and 66% a reduction of at least 50% beyond that experienced under treatment with the previously administered drug.

Chlorpromazine and Hepatic Function.

V. KINROSS-WRIGHT AND J. H. MOYER (*Arch. Neurol. & Psychiat.*, December, 1956) discuss the results of liver function tests made on patients with jaundice related to chlorpromazine therapy. These are similar to those obtained on patients with extrahepatic obstruction. Microscopically there is to be found stasis of bile in the intralobular canaliculi, with or without inflammatory infiltration. These workers investigated 30 medical and 33 psychiatric patients under treatment with chlorpromazine. They found that routine liver function tests were of no value for predicting the development of jaundice.

The Durham Test for Criminal Responsibility.

A. FORTAS (*Am. J. Psychiat.*, January, 1957) draws attention to the Durham Test as relating to criminal responsibility. The Durham test, as announced by the Court of Appeals for the District of Columbia, is: "An accused is not criminally responsible if his unlawful act was a product of mental disease or mental deficiency." Previously the test had been expressed in the substance of the M'Naghten rule, which is that to establish a defence on the grounds of insanity it must be proved that the accused was labouring under such a defect of reason from disease of the mind as not to know the nature and quality of the act he was doing or, if he did know it, he did not know that it was wrong. It is considered that the Durham principle represents a significant and desirable advance over the right-and-wrong and irresistible impulse test. The phrase "product of mental disease or defect" requires some causative relationship between the disorder and the criminal act. The Durham test does not apply in determining whether the defendant's mental condition is such that he is competent to stand trial, nor does it apply for the purposes of determining whether the defendant, who has been committed to a mental hospital because of a crime while insane, may be released. The jury may be instructed that unless they find that the evidence which establishes the criminal act was not the product of mental disease of defect, they should find the defendant not guilty by reason of insanity. Under the M'Naghten principle the example is cited of a paranoid patient who undoubtedly would know the difference between right and wrong, in which event he might be adjudged sane. It is pointed out that changes have taken place without adverse effects in our criminal system, for example, abolition of the death sentence, indeterminate sentences, and parole machinery. A closer cooperation between psychiatry and law may indicate a better method of dealing with those persons who exhibit anti-social conduct.

The Value of Tranquillizing Drugs.

P. FELDMAN (*Am. J. Psychiat.*, January, 1957) states that some people claim that tranquillizing drugs are useless, but the mass of evidence indicates they are of some value. The present study involved 1238 patients over a period of two years. The drugs used were "Thorazine" (chlorpromazine), "Pacatal", "Frenquel", and "Thorazine" combined with reserpine and "Serpasil". The trials were carried out for at least two months on the chronic in-patient population of a State hospital in which there was a high percentage of schizophrenic reactions. "Thorazine" appeared to be outstandingly superior to the other drugs tested in respect of general therapeutic efficacy. "Thorazine" combined with reserpine appeared to be the second most effective drug. "Pacatal", "Frenquel" and "Serpasil" produced significant results, but not of the same magnitude as "Thorazine". No diagnostic category seemed to respond outstandingly to any one drug. Hyperactivity, combativeness and tension states responded best to ataraxics. The response of insight, affect, judgement and realistic planning to the tranquillizing drugs is disappointing.

Temporal Lobectomy with Removal of Uncus, Hippocampus and Amygdala.

A. A. MORRIS (*Arch. Neurol. & Psychiat.*, November, 1956) reviews a series of 36 private patients three to nine years after temporal lobectomy for uncontrollable psychomotor seizures. He concludes that a completely disabled patient with medically uncontrolled psychomotor seizures, who shows a consistent unilateral electrical focus in the anterior temporal region, has a 41% chance of being free from all seizures for five years after temporal lobectomy. There is a 78% chance of being free from psychomotor seizures, a 58% chance of being free from *grand mal* seizures only, a 66% chance of attaining freedom from disturbing psychiatric complaints, and an 86% chance of gaining economic independence. Of three patients, two will have a successful operation when all practical factors have been totally evaluated. It is essential that the removal include not only the temporal tip, but also the uncus, the hippocampal gyrus and the amygdaloid nucleus.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on September 11, 1956, during the hospital annual reunion week; the medical superintendent, DR. NORMAN ROSE, was in the chair. The principal speaker was DR. K. B. NOAD, the senior honorary physician of the hospital.

Clinical History.

The following clinical history was presented.

A forty-five-year-old woman, who lived all her life in New South Wales, was married at the age of thirty-one years, and had a miscarriage at thirty-six years and no other pregnancies. She had suffered from anæmia at thirteen years and had bruised her left hip and thigh in an accident at the age of twenty-five years. Three years before her death she noticed increasing swelling of her abdomen, which was not tender or painful, and a dragging pain in the back. Her menstrual periods were irregular (and continued to be so, with long duration and heavy loss), and she thought she was pregnant. However, the lump gradually subsided, and eighteen months later she felt it only as a smaller, hard, non-tender lump. At about the same time she noticed her feet becoming numb. There were no pins and needles. Soon the left leg became weak and she was unsteady on her feet, and although the weakness gradually became worse she was afraid to seek treatment. During the last year she had lost her appetite and her weight had fallen by 21 pounds. She suffered daily headaches. Micturition was normal and there were no other symptoms. Her father (eighty years) and a sister (fifty-three years) had died of cerebral hæmorrhage. Her mother was a diabetic. The remainder of the personal and family history was irrelevant.

The patient came to hospital as a well-nourished, middle-aged woman. Foot drop was present on the left side, with wasting of the left thigh and leg (the circumference was three-quarters of an inch less than on the right side). Motor power was markedly diminished in the quadriceps and all other muscles of the left leg. The deep reflexes of the lower limb were normal and equal on both sides. There was no sensory loss. The cranial nerves were normal. An abdominal mass extended from the pelvis to six inches above and to the right of the pubic symphysis. It was attached to the uretus, irregular in contour, slightly mobile, hard and not tender. No abnormal features were noted in other systems. The blood pressure was 180 millimetres of mercury, systolic, and 90, diastolic. The urine was clear and the blood failed to react to the Wassermann and Kahn tests.

A general surgeon, a physician and a gynæcologist agreed that the pelvic mass was probably due to fibroid tumours, that there were no sensory disturbances, and that the weakness in the legs was probably caused by pressure of the pelvic growth. Two weeks after her admission to hospital the patient underwent a subtotal hysterectomy, and a firm, white tumour (17 by 12 by 10 centimetres), covered by peritoneum, was found to arise from the lower uterine segment. Examination of sections showed this to be a leiomyoma, with an unusual appearance but no convincing evidence of malignancy.

Her progress after operation was unsatisfactory. A sensory disturbance commenced in the lower extremities, and a large bedsore soon developed in the lower part of her back. The sensory loss extended till it reached the level of the tenth thoracic vertebra. Weakness and paresis of her legs gradually progressed, and she became incontinent of urine and fæces. Investigation of the spinal cord was considered, but the extensive, intractable bedsore precluded it.

Prior to her death two months after her admission to hospital she had a daily rise of temperature to 102° F., moderately severe microcytic anæmia, and neutrophilia showing a "shift to the left" and toxic changes, thought to be due to the bedsore.

Clinical Discussion.

DR. N. ROSE: I see tonight many faces which we have not seen before at a clinico-pathological conference and we wish to welcome yon, even though it is at the tail end of a long and tiring day. For those of you who do not know, I would like to pass on to you the information that these meetings have been running regularly since June, 1953. We have not missed a month. Other than this meeting, they have been held regularly on the third Tuesdays of each month; but this meeting has been brought forward a week to coincide with this reunion week. I think that you should know, too, that these meetings had been held previously over many years, but petered out about 1951, and it was due to the efforts of two people who are here today, Professor W. K. Inglis and Professor H. K. Ward, together with Dr. Malcolm Whyte, that the meetings were resuscitated and have been kept going.

Today we have an interesting case prepared by Dr. Hirst. Possibly many of you have struck such cases in your practices. Our senior physician, Dr. Noad, will open the discussion.

DR. K. B. NOAD: I hope that you have all seen the protocol. Anyhow, just in summary, this is a forty-five-year-old woman who was married at the age of thirty-one, and it was five years before she became pregnant, and that pregnancy ended in miscarriage. Subsequently she did not have any other pregnancies, and in the ensuing period of her life her menstrual periods were irregular and very heavy. Some three years before admission she began to have weakness of one leg. Before that there is a reference in the record to a lump in the abdomen, and then after that she developed weakness in the legs, some wasting of the legs and a footdrop. An operation was performed and a fibroid mass was removed and subsequently she went rather rapidly downhill with some condition which involved the spinal cord and which led to her death. That, in summary, is the history of the patient.

Now, even if we had not been told that fibroids were removed, I think that even a physician might have known that this woman had fibroids from the history. There was a long period, five years, before pregnancy, pregnancy ending in miscarriage, subsequent sterility, and throughout, irregular periods with heavy loss. All these are very characteristic of fibromyomata.

Now I usually do not criticize the case history supplied, but I was a little captious at the opening of the second paragraph. It said: "Three years before her death she noticed increasing swelling of her abdomen which was not tender or painful, and a dragging pain in the back. Her periods were irregular, and continued to be so with long duration and heavy loss. She thought she was pregnant. However, the 'lump' gradually subsided." Now what lump? Does this refer to the swelling of her abdomen? After all, one usually considers this to be a diffuse, generalized affair. Or does this refer to some tumour of which we have no details, not previously referred to, which led the lady to think she was pregnant? However, if the lump does refer to the fibroid mass which was later removed, then I cannot explain the diminution in size of this lump. I was very pleased to see Mr. Duggan come up in the lift with me, because I thought he might be able to explain it. I can only think that perhaps with the increasing size of the mass it sank into the pelvis and came to compress the nerve roots, symptoms of compression of which were noticed about this time.

Compression by the tumour is my explanation of the footdrop and the wasting of the thigh, which is mentioned in the record. In this opinion I am supported by the plurality of consultants who considered this unfortunate woman's problem. But it is interesting why there was no pain in the leg. Why was there no pain and no sensory loss? It is not referred to in the protocol. No pain and no sensory loss, but she developed footdrop and weakness and wasting of the thigh. I can only think that the major part of the compression was on the internal popliteal group which comes from the L4, L5 and S1, S2 components of the sciatic, which is motor, and which supplies the anterolateral compartment of the leg *inter alia* and would have explained the footdrop. It also takes part in the supply of the muscles of the thigh, which then would explain the mild degree of wasting of the thigh which was present. The record refers to three-quarters of an inch. That is not very much. Anything more than half an inch is perhaps significant, but anything up to half an inch is within normal limits. Three-quarters of an inch is mild wasting. So I can only think that the bulk of the compression was on that component from the lower lumbar and upper sacral segments.

Let us pass on to the events following the operation. What happened to produce the gross lesion of the cord which is evident from the events mentioned in the record? This woman began to develop numbness of the feet and rapidly developed a bedsore, a sure sign that gross interference with neurotropic function had occurred. Now, was

the pelvic tumour in fact malignant? Had it metastasized to the cord? I think that there is a good deal of evidence against that. Firstly, the histologist was not convinced that this was a malignant neoplasm. I think that the record said "no convincing evidence of malignancy". Apparently it was very doubtful if it was malignant. Secondly, if malignant change took place in such a mass, it is more likely to be a sarcoma, and such sarcomata are more likely to metastasize to viscera, particularly to the liver and the lungs. Thirdly, a malignant metastasis causing a cord compression, whether it be by involvement of the vertebræ or of the meninges, is usually an extremely painful affair before the paralysis comes on, and there is no mention of pain in the record. Finally, the symptoms and signs indicate an ascending lesion, which ultimately stopped at T10. If a metastasis developed, it is difficult to imagine that the symptoms could progress and ultimately stop at the T10 level. This is a painless paralysis and a steady paralysis. This suggests that it is due to some intrinsic condition of the cord itself.

There is a certain amount of evidence in the protocol to incriminate the arterial system in this patient. There is a reference, for what it is worth, to a family history, a father eighty and a sister fifty-three who died of cerebral hæmorrhage. Her blood pressure was 180/90 with that widening of pulse pressure which is suggestive of arteriosclerosis. However, I do not think that this woman had a thrombosis of the anterior spinal artery. Thrombosis of the anterior spinal artery is usually sudden in its onset and involves particularly the antero-lateral part of the cord. The anterior spinal artery runs down sending two major divisions into the antero-lateral compartments of the cord which are chiefly affected, leaving some evidence of function in the posterior columns. So, judging from the history and physical signs, I do not think that this was an arterial affair.

Did the woman have a myelitis? With this history of ascending paralysis, did she have a myelitis which ascended to a degree and then stopped? I think that that is unlikely. Myelitis in the ordinary sense of the term we consider to be an inflammatory affair, possibly of virus ætiology and so forth. No, I do not think that it was that. Incidentally, a person with myelitis usually has a temperature, particularly if it is in the higher reaches of the cord. A patient with a lesion at the cervical or dorsal level of the cord due to myelitis very commonly has a temperature, and might have a temperature such as they refer to here, a daily pyrexia; but I should not think that a patient with a lesion in the lower part of the cord such as this would be likely to have a temperature.

No, I think it reasonable to assume that this cord lesion was a complication of the operation. Now, there is a plexus of veins which surrounds the cord, the vertebral venous plexus, and the internal venous arrangement has communications with the large veins of the pelvis via the anterior sacral foramina. There is a good deal of evidence that some septic process was afoot, because there was a daily pyrexia to 102° F., a moderate microcytic anæmia and a neutrophilia showing a "shift to the left" and toxic changes. I think that sepsis developed in the pelvis following the operation, and that there was a spread from the pelvic veins via the anterior sacral foramina to the internal vertebral venous plexus, and that was of the nature of a septic myelitis—a septic venous thrombosis spread around the lower segments of the cord. After all, if this is so, spreading from below upwards, it is reasonable to expect that the symptoms would begin peripherally and that there would be a spread to a high level before cessation.

I think that in this patient the cause of her death was associated with a pathological lesion of the cord of a septic nature, of the nature of venous thrombosis of the cord, probably associated with the sepsis of the pelvis, and possibly associated with sepsis even within the cord. Patients subject to this condition may actually develop abscesses within the structure of the cord; but I think that it was a venous thrombosis consequent upon sepsis in the pelvis and possibly associated with sepsis in the cord itself.

DR. ROSE: Gentlemen, you have heard Dr. Noad's very clear exposition of this case, which I think has been rather cunningly selected to allow of a very free discussion from diverse quarters. Dr. Duggan, I believe, knew this case but did not do the actual operation. Dr. Duggan, I hope you are not in possession of the diagnosis; but if you are not, would you care to throw some light on this problem.

DR. A. R. H. DUGGAN: I am afraid, sir, that at a similar meeting I occupied a similar position to that which Dr. Noad occupies tonight many years ago, in regard to this very case. I was the subject picked to make a diagnosis then, so it would not be fair to comment. However, in regard to Dr. Noad's remarks about the fibroid shrinking in size, in my experience very seldom do they ever become smaller except at the menopause, when they do, and that very markedly. An ordinary fibroid does not as a rule during the reproductive life decrease very much in size.

DR. J. LANCE: Mr. Chairman, ladies and gentlemen, may I present the other possibility from the neurological point of view? If this wasting were not due to pressure of the uterine tumour, it could possibly be due to a cord lesion, to some extramedullary tumour or other lesion compressing the cord in the region of the *conus medullaris*, which would be about the level of the T12 vertebra. This patient did start off with pain in the back, though at the same time she admittedly had a developing pelvic tumour. It is possible that this pain in the back was not due to gynæcological troubles, but was in fact due to the development of some local lesion in the spinal canal. That would explain the development of the wasting of the thigh without any sensory loss and without any sphincter disturbance. If one puts it lower, in the *cauda equina*, one would expect both those things. And I think, too, that the unilaterality of her initial symptoms would make it rather difficult to explain on a local lesion of the lumbo-sacral plexus, because the tumour would have to be fairly firmly jammed into the left half of the pelvis, which is not apparent from the operative findings, and one would expect some signs on the other side even in the early stages. I think that the terminal events, exactly as Dr. Noad described, whatever we attribute the initial symptoms to, were due to thrombosis in the venous plexus in the vertebral canal with a subsequent destruction of the cord in that area.

What possible conditions in a woman of this age could cause extramedullary compression? Now, if it were a tumour, the most likely one would be a meningioma in a middle-aged woman. Neurofibroma is rare at this age. Could there be anything else, any vertebral disease possibly? We have no indication of such, but in view of one little warning sign, that this woman had lost appetite and had lost 21 pounds in weight in the last year, we could possibly think of some systemic diseases, and the one that always seems to crop up with pain in the back and possibly a subsequent paraplegia is multiple myeloma. That is always a good starter for a clinico-pathological conference, which is perhaps the most frequent place that one sees it, and I mention it as an outsider.

The other possibility that I would like to mention is an angioma, a vascular anomaly related to the cord which can remain latent for many years. In a person of middle age it may present gradually and insidiously with motor loss without sensory disturbances, and may eventually, by thrombosis occurring in the angioma, cause a paraplegia terminally.

I would therefore like to suggest that this could be a lesion involving the cord, that it could be from any of the sources of extradural compression I have mentioned, and the terminal event was thrombosis in the vertebral plexus.

DR. NOAD: Could I point out, sir, to Dr. Lance, regarding unilaterality, that it says in the protocol "about the same time she noticed her feet becoming numb". So although the signs were on one side there were subjective symptoms apparently on the other side as well.

DR. LANCE: The history stated that no sensory loss was apparent on examination, which one would perhaps rather doubt. She had been having fairly definite symptoms of numbness in both legs. This could be due to pressure on the *conus medullaris*. It does not contraindicate the possibility of a spinal lesion. The cord can become very thin as it is stretched over some extramedullary lesion, before many tracts are involved. I think that that would fit the diagnosis very well, sir.

DR. NOAD: If I might speak again, sir, I quite agree with Dr. Lance that I doubt very much that there was no sensory loss and that the reflexes were normal; but, however, we have to play the ball where it lies.

DR. W. L. CALOV: Mr. Chairman, I am afraid that I have not very many views on this. I must confess that when I looked through this, the only thing I could think of was a lesion of the cord, and the most likely thing would be a neoplasm. I admit that there are some things against it; for instance, the absence of pain, and another thing which struck me very forcibly, and which Dr. Noad had gone to pains to comment upon, is that although they say that a sensory disturbance was present in the lower extremities they do not say what kind of sensory disturbance it was, nor is there any mention of whether or not the reflexes

were exaggerated or lost, nor do they mention that there was any increase in the wasting. All these things leave me in a hazily confused state. As I said when I commenced to speak, I thought that there was primarily a lesion of the cord. Dr. Noad has made out such a good case for sepsis that my faith in my original surmise is considerably shaken.

However, there was one other feature which struck me rather forcibly—namely, that she died pretty quickly. It took her only two months to die, that is, after the surgeon had done the job. Dr. Noad has made the rather unhappy suggestion that her death might have been due to the operation, and he made out a very good case for it. I point out that there is no statement here as to the length of time that she was suffering from this pyrexia. She had a daily pyrexia, but that might have been only for a week or so before she died, and the sepsis may not have been present for a very long time, that is to say, the clinical evidence of sepsis. This makes me think that possibly there were secondaries to the tumour of the uterus; that the tumour of the uterus was after all malignant; that there were secondaries in the cord, and that there may have been secondaries in other important organs. It might have been carcinomatosis that was responsible for the woman's rapid progress downhill. I do not think that a tumour of the cord could itself be responsible for such a rapid downhill passage. If indeed there was malignancy, it must have been pretty widespread.

I still have an open mind about it, Mr. Chairman, and I do not feel like committing myself.

DR. L. BASSER: Mr. Chairman, it is quite clear that at the end of her illness the patient had a transverse lesion of the spinal cord with paraplegia, sphincter disturbance and sensory loss up to the level of the tenth thoracic vertebra. The question arises as to whether the early neurological signs were not precursors to a single clinical picture.

In regard to the question of pressure within the pelvis caused by the leiomyoma, the operation notes do not tell us how the tumour was disposed within the pelvis except to say that it was slightly mobile—that is to say, not fixed—and that it rose to the right pubic symphysis. Her earliest neurological signs were of lower motor neuron lesions on the left, so that the suspicion is raised that these signs may not have been caused by the pelvic growth. This leads to the consideration of another lesion involving the nerve roots within the spinal canal and over the distribution indicated by the wasting—that is to say, L2 to S1 on the left side. It may well be that such a space-occupying lesion grew concurrently with the neoplasm in the pelvis, and continued to grow after removal of the pelvic mass, and went on to cause compression of the cord with the final paraplegic disturbance and sensory loss.

In regard to the nature of the lesion, if the neurological story is a single one, then the history was of at least eighteen months' duration, so that one must think in terms of a benign tumour. Meningioma is quite common in women of this age. Their site is usually in the thoracic region, and in this case the lesion is at a rather lower level and, therefore, I suggest that this patient may have had a neurofibroma involving one of the nerve roots mentioned and ultimately causing compression of the cord.

DR. W. S. L. STENING: Mr. Chairman, I am rather struck by the comment of Dr. Noad's "that one must play the ball where it lies". Being an orthopaedic surgeon, I think that there are two clues that present themselves to me that might give another explanation for this patient. The first is that, being a bone surgeon, I think of lesions in bone, and the second is that there is no reference at all to an X-ray examination of the back. I rather tend to feel that if X-ray examination had been negative it would have been mentioned in the protocol, and I presume from that there might have been some demonstrable growth, and I would think that the most likely position would be that this would be a primary lesion of the vertebral body itself or the disk and would suggest as the most likely a tuberculous or infective lesion. While I will not be brought into the argument as to whether the pre-operation symptoms were due to the tumour in the pelvis or to the cord lesion, I do feel that there is a practical explanation of her deterioration after operation with a primary cord or vertebral condition in the fact that dissemination of such a lesion or actual rupture into the extramedullary space of an abscess could have been caused by the positioning of the patient on the table, and I would like to suggest that, perhaps as an outsider, a multiple myelomatosis is the cause of her condition.

DR. H. R. T. HODGKINSON: May I add one remark? There is a contradiction here, which I think you should see. Motor power was markedly diminished in the quadriceps and all the muscles of the left leg, yet we have the deep reflexes normal.

DR. G. ARTHURS: Any neurological diagnosis consists of three parts: the disorder of function, the anatomical basis of the disorder, and the pathological cause. In this case there seems to be a mixture of disorders of function with what sounds like a lower motor neuron and upper motor neuron lesion. We are told there was a footdrop on the left side, there was wasting of the thigh and leg, quite definite wasting—it was three-quarters of an inch less than the right—diminished power in the quadriceps and in the muscles of the left foot. It says that deep reflexes in the limbs were normal. It does not tell us anything about the plantar reflexes. Then, later on, she apparently had a definite upper motor neuron lesion, going up to the level of T10. Well, it is a bit difficult to combine those two in one diagnosis. She could have had some sort of tumour which caught the lower nerve roots and then climbed up the cord or, alternatively, she could have had an intrinsic lesion of the central nervous system which disseminates, such as the medulloblastoma, although she is rather old for that. But the other thing which occurred to me as a disorder of function is that this really might be something like a subacute combined degeneration of the cord, which often presents as a mixture of upper and lower motor neuron lesions.

Now the anatomical level: I do not think there is enough information. I have no idea about the pathology at all. There is one statement here which I would like to challenge, and that is that investigation of the spinal cord be considered, but the extent of the intractable bedsore precluded it. I take it that what was in mind there was a myelogram, and I would like to point out that the presence of a bedsore over a large area does not say you can not do a myelogram. By doing cisternal puncture and putting your medium in that way you get quite a good one. I have on one occasion had a case like this, a woman who was a diabetic with a diabetic neuritis and a big bed sore. She was suspected of, and in fact did have, a spinal tumour. I did a cisternal puncture and then operated just above her infected area and she did all right. No matter how hopeless the case looks, if they are a reasonable age, it is worth having a try.

PROFESSOR H. N. ROBSON: I feel it encumbent upon me, Mr. Chairman, to rise in defence of multiple myeloma and subacute combined degeneration, neither of which, in my opinion, should be considered in this case. In the case of multiple myeloma, taking the ball where it lies, I should be extremely surprised if, over a period of two months, albuminuria was not found sometimes. I suggest that on this evidence multiple myeloma could not be considered. As for subacute combined degeneration, the anaemia is desribed as being macrocytic in type, and although there are neurologists who declare that the neurological condition may occur in the absence of macrocytic anaemia, haematologically this is not generally accepted. Beyond these two impassioned statements on behalf of haematology, sir, my personal opinion of this case, for what it is worth, is that I was taught as a young man never to make two diagnoses—this, of course, possibly does not apply to clinico-pathological conferences—and would plump for a separate neoplastic lesion of the spinal cord.

Autopsy.

DR. E. HIRST: The patient had been a small, well-nourished woman weighing 83 pounds. There was notable wasting of the muscles of the left thigh. The gross findings were as follows: There was a small firm fibrous tumour 1·5 by 1·0 centimetre arising from the dura at the level of T10 and compressing the spinal cord. There was no recurrent or residual growth in the pelvis, which was normal except for a small area devoid of peritoneum. There was a large, deep, sacral bedsore. Microscopic examination of the pelvic tumour removed at operation shows appearances which resemble a neurofibroma. This appearance led to some uncertainty on the part of the pathologist. However, examination of further sections shows areas typical of a uterine leiomyoma. Dr. Tebbutt and Dr. Palmer are in agreement with this view. Section of the cord tumour shows typical appearance of meningiomatous and psammomatous meningioma. The pathological diagnosis is benign uterine leiomyoma and spinal meningioma.

Discussion.

DR. ARTHURS: May I, sir, show some X rays. These are myelograms of two cases which are both spinal tumours, but rather unusual ones. The first is taken from a young girl, aged sixteen, whom we lumbar punctured, and tipped into the completely inverted position, and she had a com-

plete block which is at T7. This sixteen-year-old girl about six weeks ago was playing some game such as netball and sat down a bit heavily on her bottom. She did not think there was much to it other than that she jarred her back, and she went on and did not think much more about that. About three weeks later she started having abdominal pains, and she had some treatment for that, but did not get any better in regard to her stomach ache. About ten days ago she began to become quite obviously paraplegic.

In retrospect it seems quite obvious that the abdominal pain she had was really root pains from some sort of pressure on her cord. The myelogram was done as an urgent matter because of the rapidly advancing paraplegia, and we immediately cut down on this lesion. When the tumour was uncapped, it was very long—about two and a half to three inches—and the upper end of it was round and smooth just like any benign tumour, purple in colour, and I thought it was probably a meningioma. But the lower end seemed to fade away into the epidural fat. There was no very definite line of demarcation, which I thought a bit queer, and I had already had my case of epidural granulomatous lesion this year, and you do not reckon on one of those more often than every three or four years. A frozen section was done and the report was that it was probably inflammatory. The pathologists saw lots of round cells, but would not definitely exclude tumour; but the opinion was that it was probably inflammatory. By that time I had removed all that I could get out, although there were a few fringes left, and I tried to stop the bleeding but could not do so.

The strange thing about this was that I could not stop the bleeding. Usually in a tumour of the epidural region you get a fair amount of bleeding, especially if it is a meningioma or something like that, but you can control it quite well if you have adequate exposure. But this just would not stop. It kept oozing away, and as we put more blood into her I thought perhaps she was bleeding because she had had so much blood put into her. However, eventually I managed to get out as gracefully as I could, having to drain it, and then a day or two later the diagnosis was told me. She had acute leuchæmia. As Professor Robson was mentioning one of these rarities this morning, I thought you would perhaps like to know about this case.

The other case is just a curiosity. This is a young boy who has had a spinal tumour shown by the myelogram, and this turned out to be a paraganglionic tumour. This was associated with hypertension, and as far as I can remember when the tumour was taken out the hypertension went away, but I believe that it is now coming back, and that this probably after all is malignant.

DR. H. M. WHYTE: I am not a pathologist, but I would like to put in another little point that the pathologists might like to throw light on, too. For the sake of truth, and for the sake of another point of interest which this case brings up, I think I might mention one fact which has been omitted, and I think it is quite in order to do so. Originally, I believe, when that uterine tumour was examined pathologically, it was reported on as being malignant, and that probably led partly to the indecision, or call it decision, to proceed no further with investigations. Subsequently, as it turned out, other pathologists reported that the tumour was not malignant, so that further investigation may have been warranted and even a cure may have been effected.

Now I remember getting a blotter through the post, as most of you may have done, which said that there was a Chinese proverb which stated: "In a dangerous illness consult three physicians." I suggest the same thing applies to pathologists.

PROFESSOR W. K. INGLIS: Mr. Chairman, this is a very old case, perhaps old enough to have been mine, because I have made so many errors in regard to uterine tumours. In point of fact it is sometimes extremely difficult to be sure whether a primary tumour of the uterus is malignant or not. But I think that Dr. Whyte's suggestion in regard to that proverb is particularly apt with tumours of the uterus.

DR. ROSE: Professor Magarey, have you any comments?

PROFESSOR F. R. MAGAREY: Only to agree wholeheartedly.

DR. ROSE: Dr. Noad, you have heard very good discussion on this case. Is there anything you would care to add before we wind up this meeting?

DR. NOAD: Only to explain, sir, that, apart from the reference to the dragging pain in the back which Dr. Lance quite correctly appreciated, and which I think is a fairly common accompaniment of tumours of the uterus, there is no further reference to pain in the whole of the record. It was that point which rather turned me off the idea of a tumour of the cord either primary or secondary. Also, there are the paucity and probably the incorrectness of the neurological data in the protocol which is no fault of Dr. Hirst. I attempted to play the ball where it lay, and so was misled.

Secondly, the evidence of sepsis in the last paragraph must, in fact, have been due to the bedsore, or probably she could have had urinary infection as well, because she was incontinent of urine and fæces. So perhaps I paid far too much attention to the suggestions of sepsis contained in the last paragraph. I quite agree with Professor Robson that it is very dangerous to make two diagnoses, so let that be a lesson to everybody.

DR. ROSE: Ladies and gentlemen, I think that you would agree that this discussion has been of very high order. It has been a very good meeting. There has been only one unfortunate incident I think, and that is this repeated reference to "play the ball where it lies". A long golf week-end has recently been concluded, and I notice several members of the audience squirming whenever this reference is mentioned. I would like you to show your appreciation, particularly to Dr. Noad and to all the others who have taken part.

Diagnosis.

Benign uterine leiomyoma and spinal meningioma.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on November 29, 1956, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, DR. EDGAR THOMSON, the President, in the chair.

The Spastic Child.

DR. R. H. VINES read a paper entitled "The Spastic Child". This paper is not available for publication.

DR. CLAUDIA BURTON-BRADLEY read a paper entitled "The Spastic Child" (see page 821).

PROFESSOR LORIMER DODS said that he was interested in the points made about the psychological problems of the parents—the need for early diagnosis and the importance of early explanation of the probable etiology. He asked whether it was Dr. Burton-Bradley's procedure to let gradually down upon the parents the full import of the child's condition, or whether she attempted to present the total picture immediately.

DR. A. R. HAMILTON said that he considered complex classifications the field of the neurologist. He stressed the value of the over-all habilitation of the spastic child, such as was carried out at The Spastic Centre in Sydney. He mentioned an operation which was sometimes valuable, and which Dr. Dennis Glissan used to perform. In that procedure all tendons round the ankle were tenotomized, with the production of a flail foot which could then be controlled by an appliance.

DR. ERIC DAVIS said that he had been associated with the work of The Spastic Centre in the field of electroencephalography for several years, and that the number of children showing electroencephalographic evidence of brain injury was most interesting. Those children attending The Spastic Centre on a daily basis were specially selected, and comprised mainly athetoid and spastic types. There were approximately only 30% of normal tracings in the spastics, but about 64% of normal tracings in the athetoids.

DR. A. R. RHYDDERCH asked whether Dr. Burton-Bradley still lengthened the hamstrings in the spastic child.

DR. C. C. MCKELLAR asked about the position of wrist fusion in the spastic. He said that he considered triple arthrodesis of value in the spastic foot in some cases. He asked whether spasticity burned itself out, as it were, since with lengthening of spastic tendons in a child the deformity often recurred, whereas in the adult it rarely recurred.

Dr. Burton-Bradley, in reply to Professor Dods, said that at the first interview with the parents of a cerebral-palsied child, she endeavoured to explain the probable cause of the child's condition; she told them that there was no cure, that the injury or defect was in the "motor" areas of the brain, and that they did not grow again, but that the child must

be helped to make the best use of his residual potentials. In all cases, the parents were given something to do. In the case of the young child, it was frequently only the constructing of a suitable posture chair. The father had satisfaction in making (or organizing the making of) the chair, and the chair itself allowed the child to sit up and look at the world around him. Dr. Burton-Bradley tried to give them the philosophy of being pleased with each little gain, while they retained hope for further improvement. In reply to Dr. Hamilton, Dr. Burton-Bradley said that she considered that classification into the main types of cerebral palsy should be understood by orthopædic surgeons, and that it was particularly necessary for them to differentiate between the spastic type and the athetoid type. It was also important for them to be able to assess in the spastic type which individual muscles were spastic, and which were normal, weakened or flaccid. Failure to understand muscle imbalance in the spastic was frequently the cause of the "failure" or poor result in many patients subjected to operation. She had not used Dr. Glissan's operation. In answer to Dr. Rhydderch, Dr. Burton-Bradley said that very occasionally in selected cases she lengthened the hamstrings. In answer to Dr. McKellar, Dr. Burton-Bradley said that the position of wrist fusion was the one of election by the patient, and that the procedure was carried out only in the adult. The wrist was placed in the position that the patient found most useful, and was kept in that position in plaster for three months, during which time the patient used the hand. If at the end of that time he wanted that position maintained, fusion as nearly as possible in that position was carried out. Dr. Burton-Bradley said that triple arthrodesis had proved of value in the young adult in some cases. The answer to spasticity "burning out" appeared to be in the problem of growth. The spastic muscle did not grow at the same rate as normal muscle. The presence of a normal agonist with a spastic antagonist or vice versa would result in contraction of the spastic muscle, which would be worse as the child grew. Operation during the rapid growth period would reduce the hyperirritability for a while; but the muscle was still spastic, and would not grow at the same rate as its agonist, so the condition relapsed if the limb was not held by an appliance. In the adult, the growth period was over.

A MEETING of the Victorian Branch of the British Medical Association was held on March 6, 1957, at the Footscray and District Hospital, Melbourne. The meeting took the form of a series of clinical demonstrations by the honorary medical staff of the hospital.

Trauma of the Upper Extremity.

DR. CONRAD LEY showed a girl, aged seven years, who had been first examined on March 29, 1956. At that time she had a right ulnar nerve lesion, apparently due to an injury which she had sustained by falling into a glass window about three months previously. On April 17 exploration was undertaken, and resuture of the nerve and transposition were carried out. On September 18 there was some return of sensation over the distribution of the nerve, but only slight motor improvement, if any. Eleven months later, at the time of the meeting, she had almost complete return of ulnar nerve function.

Dr. Ley's second patient was a man, aged sixty years, who had sustained an injury to his right elbow, due to its protruding whilst he was driving. He was admitted to hospital on September 13, 1955, with a mangled right elbow and a fractured right clavicle; the main vessels and nerves were intact. Operation was undertaken. First, *débridement* of muscle, bone, skin, fragmented humeral condyles, the proximal third of the ulna, the fractured middle third of the radius, the disorganized elbow joint and the "degloved" forearm was carried out; then an intramedullary wire was inserted through the radius and skin was grafted. Later on further grafting was carried out, and the patient returned to work in March, 1956. He was then given a stabilizing splint to counteract the excessive joint mobility.

The next patient shown by Dr. Ley was a man, aged twenty-two years, who had sustained a fracture of the right humerus with radial nerve palsy. He had been admitted to hospital on May 26, 1955, when an open reduction of the fracture was performed. Because of non-union, a bone grafting procedure was carried out on February 22, 1956. On July 5, nerve exploration and suture were undertaken (approximately fourteen months after the original injury). At the time of the meeting there was no return of radial nerve function. Dr. Ley said that the outlook was poor, on account of the lapse of time between the original nerve injury and the time of excision and suture. The patient was actively employed, wearing a radial nerve splint. A further period of nine to twelve months would elapse before tendon transplantation was contemplated.

Dr. Ley finally showed a male patient, aged thirty years, who on December 6, 1956, had sustained a traumatic amputation of the right forearm through being caught in a belt at work. At operation the skin and vessels were trimmed, a three-inch stump being left. On February 7, 1957, the stump was well healed, and the patient was awaiting a prosthesis.

Backache as Seen in the Out-Patient Department.

DR. BRUCE R. HALLOWS discussed backache as seen in the out-patient department. He said that from early antiquity man had been complaining of backache. Ever since man adopted the erect posture, he had had to combat the ceaseless force of gravity. To that end, he had developed the large, powerful antigravity muscles, and the highly efficient stretch reflex. The problem of backache was one of posture, and in attempting to elucidate it, one had to consider the complex nature of the spinal column and its associated muscular, ligamentous and neurological components. The problem of backache as seen in the out-patient department represented a good cross-section of similar cases in a busy general practice. From the practical and economic points of view, it was becoming of increasing importance, especially in relation to workers' compensation cases. To realize how important that was, one had only to remember the tragic incident in Brisbane that had occurred some little time previously. The problem produced three questions: (*a*) from the patient, what was the cause of his pain and whether it could be cured; (*b*) from the employer, how long the patient would be away from work, and whether he would be able to carry on his former occupation and at the same *tempo*; (*c*) from the doctor, how he might fulfil the requisites of the previous two questions.

Dr. Hallows went on to say that for the purposes of simplicity, backache might be divided into two main classes—very serious causes, and less serious but important causes. The very serious causes comprised the following nine categories: (i) Neoplasms, which could be primary tumours (angioma, osteoclastoma) or else secondary metastases. The primary tumours were rare. The secondary metastases were relatively common, the primary lesion being in the prostate, breast, thyroid, stomach or other parts of the body. (ii) Myelomatosis. (iii) Paget's disease. The pain was usually due to concomitant arthritis. (iv) Tuberculosis. The rigidity of the spine, the root pains, the association of a pulmonary lesion, and a positive response to the Mantoux test established the diagnosis. (v) Spondylolisthesis. The forward movement of the upper vertebral body usually took place at the lumbo-sacral junction. The underlying pathology was a congenital defect of the *pars articularis*. If the defect was present without any appreciable slip, it was known as spondylosis. (vi) Ankylosing spondylitis. The early radiological signs appeared in the sacro-iliac joints, and the erythrocyte sedimentation rate was raised. That condition was suspected in young males who complained of low lumbar backache, and when the pain radiated to the gluteal regions. (vii) Osteoporosis. The backache might be "lumbago" due to sudden fracture or collapse of a vertebral body. Idiopathic vertebral osteoporosis occurred in middle-aged women and gave rise to backache. (viii) Fractures. Fractures might be of the compression type, fracture of the body of a vertebra, or of the transverse or spinous processes. It was the common experience that in fractures of the spine the site of the pain was not usually at the fracture and was almost certainly due to tearing of the neighbouring soft tissues. (ix) Rare conditions. Among rare causes of backache were osteomyelitis and erosion from pressure of an aneurysm.

Dr. Hallows then discussed the less serious but important causes, and classified them under the following five headings: (i) Trauma. Strains from bending or lifting and heavy falls might cause compression of the lumbar intervertebral disks, tearing of the lumbar muscles or their attachment to the pelvis, tearing of the interspinous or other ligaments, or contusion of the articular cartilage of the facet articulations. There was a history of pain at the onset, but that frequently gave way to a chronic ache in the lumbo-sacral region, as the result of stretching of scar tissue. (ii) Osteoarthritis of various articulations. That commonly caused pain even before X-ray changes could be shown. It was the arthritis of the facet articulations that was really important. To show that, special oblique views were necessary. Osteophytes might cause symptoms of

compression of nerve roots. (iii) Prolapse of an intervertebral disk. That was usually preceded by some degenerative process, and frequently an accident or injury might act as the trigger focus and initiate the symptoms which followed. As the disk was extruded the cushion effect between the bones was lost, and then followed abnormal stretching of the ligaments and capsule of the facet articulations. That stage was responsible for the backache which preceded the sciatica as the result of compression by the disk on the component roots of the sciatic nerve. (iv) Postural strains, such as those caused by excessive weight, weakened muscles after illness, poor posture and occasionally pregnancy. They all caused pain as a result of the stretching of ligaments, and the continued muscular effort made in an attempt to rectify the abnormal posture. (v) Rheumatism. Frequently one might elicit a previous history of joint pains in other parts of the body as well as past attacks of fibrositis elsewhere, such as the "stiff neck" or the painful shoulder.

Dr. Hallows then said that the symptomatology was as diverse as the number of pathological causes. However, some fairly well-defined syndromes had become apparent. The first was backache associated with trauma. In that one found the history of an accident, with immediate onset of pain, associated exacerbation of pain during particular movements (such as coughing, bending or turning), limitation of movement, and a localized area or localized areas of tenderness. In those cases it was absolutely necessary to make a careful X-ray examination, to determine whether there was an osseous lesion, such as a fracture of the body, or of the transverse or spinous processes. The second syndrome was backache without trauma. Under that heading were included those cases in which backache followed work done in unnatural positions or necessitating bending for prolonged periods (for example, such patients might be surgeons, dentists or mechanics working over a bench). Postural deformities, obesity and illness also produced that type of backache. Presumably the underlying pathology was stretching of ligaments, with muscular effort to counter the stretching. The third syndrome comprised disk lesions. They were usually characterized by a history of injury (though not in every case) followed by backache for some varying period of time, and then the development of pain radiating along the distribution of the sciatic nerve—in the buttock, down the back of the thigh, along the outer side of the calf and even down to the outer side of the ankle. With that might be noticed the wasting of the calf muscles, diminished or absent ankle jerk, and on occasions the complaint of numbness and/or "pins and needles" along the course of the sciatic nerve. Sensory loss was not common, but it did appear. Leg raising tests and the like usually confirmed the signs and symptoms enumerated. In the absence of osseous lesions and signs of gross neurological or cord disorders, it was safe to say that an extruded disk was the most likely cause of such symptoms. The third category comprised backache of divers natures. Chronic low backache might be the result of osteoarthritis of the spinal articulations. It was of a more or less chronic form interspersed with more acute attacks, and often associated with a history of arthritis in other parts of the body. It was also found in cases of "lumbago", which were characterized by a sudden attack of pain in the lower lumbar region following some trivial movement, such as bending down or getting out of bed or having a shave. Then there was the backache of lumbar fibrositis with the localized nodule of tenderness (the "multifidus" syndrome), and the chronic aching pain of generalized osteoporosis had to be remembered. In the fourth category also were placed those cases of backache which followed sitting in draughty places, and there was little doubt that such cases did occur.

Dr. Hallows then discussed investigation. He said that it was of paramount importance that (i) an accurate, chronological history should be taken, (ii) a careful clinical examination should be made, and (iii) an X-ray examination should be made. The clinical examination was made by inspection of the back, with particular attention to the presence of (*a*) a flattening of the lumbar curve or a scoliosis, (*b*) limitation of movement (forward bending, backward extension), (*c*) localized tenderness in lateral muscle masses or few or numerous small localized areas of tenderness, (*d*) any wasting of the calf muscles, and (*e*) any alteration in the ankle jerk. If any suspicion arose, there was need for a careful examination of the pelvis and rectum, and finally for an examination of the urine, as bilateral sciatica was often the result of *diabetes mellitus*. X-ray examination was of great value. It not only revealed serious lesions, but frequently the report was "a mild scoliosis with a slight narrowing of the disk space between L5 and the sacrum". That report, if associated with a clinical history of radiating pain, was most suggestive of a disk lesion. The question of myelography was still *sub judice*.

Referring to treatment, Dr. Hallows said that the treatment of backache was that of the cause, and the lack of a correct diagnosis was frequently the explanation of the poor response to treatment. In the acute phase following an injury, rest in bed and the exhibition of an analgesic were of great benefit. After that phase had passed, remedial exercise, which strengthened muscles and stretched adhesions and contracted capsules, should be instituted. For the patient with "lumbago" and the fibrositic nodule, infiltration with a local anæsthetic agent was of great benefit. In that connexion Dr. Hallows stressed the necessity for accurate localization of all the tender nodules to give complete relief. He thought that if a long-acting, safe local anæsthetic was available, it would be of great value. In the true rheumatic cases, there was little doubt about the value of "Butazolidin"; but it must be used with care. Plaster jackets were of value in the management of compression fractures of the vertebral body, after the fracture had been adequately reduced, and in some cases of osteoporosis associated with chronic backache, a spinal support had been found valuable. With regard to manipulation, Dr. Hallows said that it had three possible effects: (*a*) to replace displacements, (*b*) to break down adhesions, and (*c*) to stretch contracted capsules of an injured or osteoarthritic joint. It had been found useful in lumbago, in sacro-iliac strain and in disk lesions. In the treatment of disk lesions, it had been the custom at the hospital to manipulate the patients under general anæsthesia, and then to examine them in two or three days' time. Frequently they said that their sciatic pain has disappeared or decreased, but they then complained of a fresh pain, usually in the region of the posterior superior iliac spine, and that area was then infiltrated with a local anæsthetic agent. In a number of cases that regime was proving satisfactory. Operative procedures were reserved for prolapsed intervertebral disk lesions which had not responded to any form of treatment, and for other serious lesions such as spondylolisthesis. Dr. Hallows finally mentioned the use of non-specific vaccines for the unusual type of backache which might be an expression of some latent septic focus.

Urethral Injuries.

DR. I. H. OGILVY showed two patients illustrating the management of urethral injuries. The first patient, a boy, aged ten years, had been admitted to hospital on January 22, 1954, after being struck by a car. There was a large wound in the left inguinal region, and the external iliac vessels, the femoral nerve, and the flexor muscles of the hip joint had been severed. The left leg and foot were cold and cyanosed, and no pulses were palpable. There were extensive lacerations of the scrotum with considerable skin loss, and the left testis and cord had been avulsed. The right testis and cord were exposed, but otherwise uninjured. The skin of the *glans penis* was intact, but the skin of the shaft of the penis had been completely avulsed, together with the urethra and *corpus spongiosum*, from the base of the glans to the peno-scrotal junction. In addition, there were multiple small lacerations and abrasions over the lower part of the abdomen and both thighs. Radiological examination demonstrated the presence of fractures of the body of the left pubis and of the left iliac crest. Shock was severe, but responded to blood transfusion. At operation the external iliac vessels were ligated. Repair of the femoral nerve was not possible, as a gap of two inches was present. The wounds of the left inguinal region and the scrotum were sutured, and the right testis was replaced in the remaining part of the scrotum. A split-skin graft was applied to the shaft of the penis, and a suprapubic cystostomy was established. On January 29 obvious gangrene of the left leg below the mid-calf was present. As no recovery of the *quadriceps femoris* muscle was possible, amputation above the knee appeared necessary, but was deferred owing to extensive skin damage to the thigh at the time of the accident. Thus, on January 29 amputation below the knee was carried out, and was followed by amputation through the lower part of the thigh on April 29. At that time the large wound of the left inguinal region, which had broken down because of infection, was healed. The skin graft to the penis took satisfactorily, and the suprapubic tube was removed on February 18. The patient was discharged home on crutches in May, 1954.

In August, 1954, reconstruction of the penile urethra was carried out in the following way. Two parallel skin incisions, a quarter of an inch apart, were made on the undersurface of the penis between the base of the glans (in which the

urethra was intact) and the site of the urethral opening in the perineum. A flap of skin and fat, with its base just behind the posterior urethral opening, was raised from the perineum to one side of the mid-line. That flap was then rotated into the mid-line and sutured to the outer edges of the skin incisions on the undersurface of the penis over a number 18F Foley catheter. In that way the continuity of the urethra was restored, the roof being formed by a strip of skin on the undersurface of the penis, and the floor by the flap of fat and skin, the principle being similar to the Denis Browne method for repair of hypospadias. A split-skin graft was applied to the site from which the flap had been raised. Healing proceeded by primary intention except at the apex of the skin flap, where some separation of the suture line occurred, and a small fistula persisted at that site. The catheter and sutures were removed on the fourteenth post-operative day.

The fistula at the base of the glans persisted, and in January, 1955, an attempt at closure over an indwelling catheter failed. A stricture was present at the base of the glans just distal to the fistula, but the latter remained patent in spite of repeated dilatation of the stricture. In June, 1955, the patient was readmitted to hospital. The urethral stricture was again dilated, a suprapubic cystostomy was established, and the fistula was repaired by means of a small rotation flap. On that occasion healing of the fistula occurred, and the suprapubic catheter was removed on the twenty-third post-operative day. Two days later the patient was passing urine via the urethra without difficulty, and there was no passage of urine through the suprapubic wound.

In January, 1956, the patient was able to flex the left hip through 90°, a surprising range of movement in view of the extensive muscle damage which had occurred initially. An artificial limb was supplied, and he learned to use it satisfactorily in a short time. There was no dysuria, and repeated dilatation of the urethra had not been necessary. The right testis was situated in the scrotum, and was developing normally.

Dr. Ogilvy's second patient was a married man, aged twenty-six years, who had been admitted to hospital on August 1, 1954, after an accident in which he had been struck by a motor-car. Urine had not been passed since the accident. Severe shock was present, and there were tenderness and rigidity over the lower part of the abdomen, and pain on pressure over the pelvis. In addition, there was a wound two inches long in the right perineal region, and rectal examination revealed upward displacement of the prostate gland. Radiological examination of the pelvis demonstrated fractures of both right and left superior and inferior pubic rami with downward displacement of the pubis. There was also a vertical fracture through the right side of the sacrum with some upward displacement of the right side of the pelvis. Other injuries included a fracture of the right clavicle and probable rib fractures.

The shock responded to blood transfusion, and early operation was performed. Catheterization produced a few ounces of blood, but no urine was obtained. A mid-line suprapubic incision was made, and the bladder was opened. The latter was intact, and the diagnosis of a complete rupture of the membranous urethra was confirmed. Metal sounds were passed along the urethra from each site of the rupture, and the lower sound was then manipulated across the rupture and through the prostatic urethra into the bladder. A 24F Foley catheter was then withdrawn from the bladder, through the urethra, over the end of the sound. The wound was closed, with suprapubic drainage of the bladder, and the perineal wound was sutured. Post-operatively, penicillin, streptomycin and sulphonamide were given. Convalescence was complicated by the development of gross surgical emphysema as a result of the chest injury. Chemotherapy was discontinued after ten days, and the urethral catheter was removed after four weeks. At that time urethral sounds were passed without difficulty, and the suprapubic tube was removed. Six weeks after operation the suprapubic wound had healed and there was no dysuria. On September 27 sounds were again passed without difficulty. At that time the patient was walking a little and was discharged home.

Dr. Ogilvy said that since the accident there had been no dysuria, although the urinary stream was somewhat smaller than it had been prior to injury. Repeated dilatation of the urethra had not been necessary, and the degree of stricturing was minimal. For many months after the accident penile erection was absent and still occurred only incompletely, but the patient's first child had been born in 1956.

Fractures of the Arm and Forearm.

Dr. Ogilvy next showed two patients who had sustained fractures of the arm and forearm in motor-car accidents. The first patient, a male, aged thirty-seven years, had been admitted to hospital on January 15, 1955. He had been thrown out of a truck after a collision, and had sustained a compound supracondylar fracture of the right humerus. There was a grossly contaminated wound, two inches in length, on the lateral aspect of the elbow, and bone was protruding. Radiological examination revealed what was essentially a supracondylar fracture with a vertical extension into the elbow joint, but gross comminution of the fragments had occurred. At operation *débridement* of the wound was performed, and one contaminated fragment of bone was removed. Some improvement in the position of the fragments was obtained, the wound was sutured and a padded plaster cast was applied. Chemotherapy was commenced prior to operation. Post-operative X-ray examination showed that the position had deteriorated, and on January 24 further manipulation was carried out, but resulted in only temporary improvement owing to the instability of the fragments. The patient was discharged home on January 26 with the arm in a plaster cast.

Seven weeks after injury the plaster cast was removed, and clinical and radiological union were found to be progressing satisfactorily. The wound was soundly healed, and active exercises were commenced. In June, 1956, a 45° range of movement had been regained. However, a small sinus developed in the wound, and over the succeeding four months three small sequestra were discharged. Healing of the sinus then occurred, and radiological examination revealed no evidence of active osteomyelitis or further sequestra. The patient had resumed his work as a golf-club maker in April, 1955, and was able to manage satisfactorily. He finally regained a 60° range of painless movement—a surprising recovery in view of the severe comminution of the fracture, the involvement of the elbow joint, and the occurrence of infection.

Dr. Ogilvy's next patient, a male, aged twenty-two years, had been admitted to hospital on April 7, 1956. He had sustained a supracondylar fracture of the humerus with backward displacement of the distal fragment, a compound fracture at the junction of the upper and middle thirds of the shaft of the ulna with anterior bowing and anterior dislocation of the head of the radius (Monteggia fracture), and a further fracture of the distal shaft of the ulna without significant displacement. Immediate operation was carried out, and the proximal fracture of the ulna was reduced and plated. The supracondylar fracture and the head of the radius were reduced by manipulation, and a plaster cast was applied. However, further X-ray films taken on April 10, 1956, showed that redislocation of the head of the radius had occurred, and that was again reduced by manipulation. The plaster was finally removed twelve weeks after injury. At that time the supracondylar and distal ulnar fractures were soundly united in satisfactory position, and reduction of the head of the radius had been maintained. Union of the proximal ulnar fracture was incomplete, and slight distraction was present. In August, 1956, practically full movement had been regained, and the patient resumed work as a cleaner.

Dr. Ogilvy said that on January 21, 1957, full movement had been regained, and there were no symptoms except for occasional aching of the forearm. However, X-ray films showed that union of the proximal ulnar fracture had not occurred. A bone graft would be necessary to obtain union of the fracture.

Carcinoma of the Ureter.

Dr. Ogilvy finally showed a male patient, aged seventy-three years, who had first attended the Footscray and District Hospital on September 26, 1954. Eight months previously the patient had noticed blood in his urine after receiving a blow in the right inguinal region. Endoscopic examination had revealed no abnormality of the bladder, and no further investigations had been carried out. Hæmaturia recurred on September 22, 1954, and the patient was admitted to hospital four days later with clot retention. The prostate gland felt normal on rectal examination. The retention of urine was relieved by catheterization, but severe hæmaturia continued, with the passage of clots. Endoscopic examination on the day following his admission to hospital revealed that blood was emerging from the right ureteric orifice, and that the bladder itself was normal. In view of the severity and persistence of the bleeding, immediate operation was proceeded with, and the right kidney was exposed through a loin incision. The kidney was found to be hydronephrotic,

and on palpation of the ureter, a small mass was felt approximately three inches above the bladder. The peritoneum was opened, and the left kidney was found to be present and of normal size. A right nephro-ureterectomy was performed, the lower end of the ureter being exposed by an extraperitoneal approach through a right paramedian incision. The lower two inches of the ureter appeared normal, and the lower end was divided flush with the bladder.

Macroscopic examination of the specimen showed gross hydronephrosis of the kidney, but no growth was present in the pelvis. A papillary growth was present in the ureter, and subsequent microscopic examination showed this to be a transitional cell carcinoma with invasion of the muscle coat and adventitia.

A wound abscess was drained four weeks after operation, and the patient remained well until nineteen months after operation. At that time hæmaturia recurred, and endoscopic examination revealed a papillary growth confined to the site of the right ureteric orifice. The area was treated with diathermy, and since that time repeated diathermy had kept the recurrent growth under control. No mass was palpable on abdominal examination, and there was no evidence of distant metastases.

The Value of Biopsy of Common Superficial Lesions.

DR. W. B. FLEMING showed several patients to illustrate the surprises one might receive if apparently simple superficial lesions were as a routine sent to a pathologist after removal. An apparently infected sebaceous cyst was found to be a superficial sarcoma; another small lump removed from a forearm was found to be an isolated manifestation of sarcoidosis. One other patient shown had a melanoma on the sole of the foot, which had been treated with silver nitrate applications for some weeks, because it was thought to be a plantar wart. Another patient was shown to illustrate the extent of the resection necessary when an excisional biopsy of a suspected melanoma was performed.

Varicose Veins and Varicose Ulcers.

DR. J. KENDALL FRANCIS presented three patients, to demonstrate some of the complicating factors in choosing the correct treatment of varicose veins and ulcers.

This first patient was a married woman, who had presented with a right inguinal hernia, but who also had associated bilateral varicose veins of the internal saphenous systems. Along the line of the saphenous veins on both sides were several incompetent communicating veins or "blow-outs". Tourniquets applied to prevent retrograde reflux of blood down the saphenous veins failed to prevent filling of the "blow-outs". The "blow-outs" near the medial malleolus could be felt emerging through foramina in the deep fascia, which were marked on the skin before operation. At operation (the findings being similar on both legs) the Trendelenburg ligation in the groin of the internal saphenous vein and its tributaries was performed, after which the saphenous vein was exposed at the ankle and the intraluminal stripper was passed from ankle to groin with ease. The stripper was left in position, and the "blow-outs", which were seen to lie close to the line of the stripper, were then exposed by small transverse incisions—two incisions above the ankle on the medial side, and one incision over Hunter's canal in the mid-thigh region. The incompetent perforating veins were found to be separate and not in any direct communication with the long saphenous trunk itself; undoubtedly, those would have been the cause of recurrent varicose veins if merely a full stripping operation had been performed and the "blow-outs" had been left. The "blow-outs" were ligated where they emerged through the deep fascia, and the transverse incisions were then closed. The stripper was then pulled through from below upwards, whilst elastic bandages (ascending behind the stripper) were firmly wound round the leg to prevent hæmatoma formation. The transverse incisions healed admirably. Dr. Francis said that they were more suitable (in cases in which the "blow-outs" could be localized clinically) than the long vertical incision behind the medial border of the tibia recommended by Cockett.

Dr. Francis commented that the patient under discussion would undoubtedly have developed sequelæ of those incompetent perforating veins—namely, increasing œdema, pigmentation, induration and finally ulceration—if they had been left. It was important, therefore, to look for and ligate such "blow-outs" separately, as stripping of the saphenous vein would not remove those incompetent communicating veins.

Dr. Francis's second patient was a man, aged fifty-six years, who had presented with an indolent ulcer overlying his left medial malleolus, which had been present since he had received a knock over that area four months previously. Despite rest in bed and the application of dressings, the ulcer had adamantly refused to heal. The patient had associated bilateral varicose veins of long standing and had been regarded as suffering from a purely varicose ulcer. However, the reason for the failure of the ulcer to heal was found on closer questioning, when the patient gave a classical story of intermittent claudication and of ischæmic rest pain in that leg. On examination of the patient, he had gross bilateral varicose veins of the long saphenous systems, with several "blow-outs" in close relation to an indolent ulcer over his left medial malleolus, the adjacent skin being thin and pigmented. Both feet were ischæmic, and no pulses were palpable below the femoral artery on either side. Dr. Francis said that that was a not uncommon problem—the coexistent roles of ischæmia and venous insufficiency in the causation of malleolar ulcers—and treatment might be difficult. Attempts to increase the arterial inflow further burdened the already overloaded venous efflux, whilst a compression regime to control the dilated veins might, if incorrectly applied, cause further ischæmia of the foot owing to arterial compression. In the case under discussion, the ulcer was healed by the judicious use of well-padded compression bandages, reapplied weekly, which was followed up by a left lumbar sympathectomy to alleviate the rest pain and attempt to maintain the ulcer in its healed state. The results from both points of view were excellent. It might well be that that was the ideal case, in which lumbar sympathectomy played a major role in the healing of a primarily "varicose" ulcer, enabling firmer compression bandages to be applied with less risk of causing distal ischæmia. The venous insufficiency was subsequently controlled by well-fitting elastic stockings, as surgical attack on the veins seemed too fraught with the risk of wound-edge necrosis in most such cases.

Dr. Francis finally showed a married woman, aged sixty-three years, who had presented with gross bilateral varicose ulcers, which she had had for over twenty years; they had first occurred ten years after a bilateral post-partum deep venous thrombosis. She had also experienced severe Raynaud's vasospastic attacks in all four limbs for the last eight years; the spasm and cyanosis were almost continuous during the cooler weather, and resistant to all forms of therapy. A further association was mild bilateral varicose veins. The ulcers had recurred repeatedly despite many lines of therapy—namely, multiple ligation of the varicose veins, repeated skin grafts and periods of rest in bed, compression bandaging regimes and, finally, a left lumbar sympathectomy with further skin graft. All were of no avail; the ulcers recurred on both legs, although the Raynaud's attacks had become less severe in the sympathectomized limb. Ascending venography revealed tortuous deep veins, but the valves were competent in the popliteal vein—no "blow-outs" could be shown. Treatment consisted of rest in bed and the application of dressings for a week until the ulcers were clean; they were then grafted with Thiersch grafts, which "took" admirably. Three weeks later the grafts seemed quite firm, and exploration through one of the previously grafted sites was undertaken in an endeavour to show and ligate the incompetent communicating veins believed to be the basic cause of the ulcers. No true "blow-outs" were found—merely small competent perforating veins and tortuous posterior tibial *venæ comitantes*; they were ligated and the wound was closed. Wound edge necrosis occurred to a moderate extent, owing to considerable undermining of the grafted area at operation during the search for "blow-outs". It healed in a few weeks, but demonstrated the inadvisability of too early and too extensive an operation through the grafted skin. No further surgical treatment was undertaken for the two other healed ulcers. At the time of the meeting the patient, by wearing well-fitted elastic stockings, had prevented any recurrence of the ulceration for over twelve months, and the grafted skin over the old ulcer sites had thickened and become almost as supple as the adjacent normal skin. Raynaud's attacks were still frequent and were her most distressing complaint.

Dr. Francis said that the patient demonstrated again the problem of associated venous insufficiency and ischæmia. In the present case the latter was of a spasmodic nature, but nevertheless he thought that it predisposed an already abnormal venous drainage to further handicaps, and militated against healing of the "varicose" ulcers once they had occurred.

Compression Stockings.

Dr. Francis demonstrated the advantages and disadvantages in several of the common types of compression stockings in use. He said that the long one-way-stretch strong elastic bandage applied daily by an intelligent patient might accelerate healing of many ulcers resistant to other measures, at the same time greatly lessening the time spent by the patient in travelling to and from hospital, and by the medical officer and the nursing staff in applying other supportive regimes.

Recurrent Rheumatic Fever.

DR. N. L. DODD showed a male patient, a quarryman, aged thirty-four years, who had been admitted to hospital on January 31, 1957, with a history of polyarthritis and fever of three weeks' duration. He had had chorea at the age of five and seven years, rheumatic fever at twenty-four years, and no other serious illness. Three weeks before his admission to hospital he had complained of pain in both shoulders, more severe in the left than in the right. One week before his admission he had developed pain in both hips, and pain and stiffness in his fingers and toes. Two days before his admission he had developed pain in the left knee. He had felt feverish for one month, and had had a slight, unproductive cough for a week.

On his admission to hospital, the patient's temperature was 98° F., his pulse rate was 96 per minute and regular, and his respirations numbered 24 per minute. His blood pressure was 180 millimetres of mercury, systolic, and 100 millimetres, diastolic. The apex beat was in the fifth left intercostal space five inches from the mid-sternal line. A regular dual rhythm was present. A presystolic murmur and a rumbling diastolic murmur were heard at the apex, and there was a blowing systolic murmur in the aortic area. Joint movement was limited by pain. The fingers were swollen at the proximal interphalangeal joints, and flexion was limited; the toes were tender, and movement was limited. The reflexes were equal and active; the plantar reflexes were flexor in type. The erythrocyte sedimentation rate was 117 millimetres in one hour, and the hæmoglobin value was 76%.

The patient was given sodium salicylate in a dosage of 20 grains every four hours for two days, and then 20 grains every six hours. The pain gradually diminished in his joints, but they remained rather stiff, with some limitation of movement; there was weakness of the intrinsic muscles of the hands. Treatment with prednisolone (five milligrammes twice a day) had been started two weeks prior to the meeting, and also physiotherapy in the form of exercise and the application of heat. Considerable improvement in the range of joint movements had resulted.

Polyarthritis.

Dr. Dodd next showed a male patient, aged forty-eight years, a cotton dyer, who had been admitted to hospital on February 5, 1957, with polyarthritis of one day's duration. He had had no previous serious illness. One month before his admission to hospital he had developed a sore throat, with fever, malaise and headache. His condition improved with penicillin treatment. Two weeks before his admission to hospital he was examined by his doctor; his temperature was then 99·4° F., his pulse rate was 90 per minute, and his blood pressure was 140 millimetres of mercury, systolic, and 90, diastolic. There was a fine purpuric rash on the upper part of the chest and shoulders, and a sparse maculopapular rash was present over the same area. The urine was acid, and contained no albumin or sugar; microscopic examination of the uncentrifuged deposit showed it to contain ten leucocytes per low-power field. A blood examination on January 31, 1957, gave the following information: the hæmoglobin value was 83%, the erythrocytes numbered 4,000,000 per cubic millimetre and the leucocytes numbered 7000 per cubic millimetre; in a blood film, erythrocytes were normal and platelets numerous. The patient was well when he was examined on February 2, with a temperature of 98·6° F. On the day before his admission to hospital he developed arthritis with effusion in both wrists, and arthritis of both elbows, both ankles, both knees and both hips.

On his admission to hospital the patient's temperature was 100·2° F., his pulse rate was 104 per minute and regular, and his respirations numbered 20 per minute. His blood pressure was 128 millimetres of mercury, systolic, and 80 millimetres, diastolic. No abnormality was detected in his heart. Joint movement was limited by pain. The erythrocyte sedimentation rate was 105 millimetres in one hour, and the hæmoglobin value was 72%.

The patient was treated by rest in bed, and the administration of sodium salicylate in a dosage of 20 grains every four hours for two days, and then every six hours. His temperature remained high, and there was only slight improvement in the affected joints. Three days after his admission to hospital, cortisone therapy was started; 300 milligrammes were given on the first day, 200 milligrammes on the second day and 100 milligrammes per day thereafter. Joint pain disappeared overnight, and a full range of movement was reestablished in all joints. His temperature returned to normal. Dr. Dodd said that the patient had continued to feel well ever since, and was now having 75 milligrammes of cortisone acetate per day. There had been a very big reduction in the erythrocyte sedimentation rate.

Disseminated Lupus Erythematosus.

Dr. Dodd's third patient was a young woman, aged nineteen years, an accounting machine operator, who had been admitted to hospital on October 25, 1956, with a history of polyarthritis of eight weeks' duration. She had had acute nephritis at the age of ten years, and had suffered from pain in the right iliac fossa ten months and two months before her admission to hospital. Eight weeks before her admission to hospital she had developed painful swellings in both knees, which persisted for two days and then subsided. Later, the pain migrated from joint to joint, involving the finger joints, the right wrist, both elbows, both shoulders and the left ankle. On her admission to hospital the left knee and both elbows were affected. She had shown no response to salicylates up to that time. Two weeks before her admission she had had a severe cold, with pain in the left upper part of the chest; and one week before her admission she had been febrile and sweating for three days. At that time she also had diarrhœa, and noticed slimy material in her bowel actions, but no blood. She had also been anorexic for six weeks, and had lost weight. An upper tooth had been extracted four weeks before her admission to hospital. She had had a slight cough for two weeks. Micturition and menstruation were normal.

On examination of the patient on her admission to hospital, her temperature was 102° F., her pulse rate was 100 per minute, and her blood pressure was 120 millimetres of mercury, systolic, and 70 millimetres, diastolic. An irregular, raised rash was present on the forehead and cheek; the fauces were reddened. Breath sounds were diminished low in the left axilla. Coarse râles were heard at the end of inspiration over the left lobe anteriorly. Vocal resonance was normal. No abnormality was detected in the abdomen. The left knee was swollen and hot, and movement of the elbows was painful. A small left tonsillar node was palpable, and there were palpable nodes in both axillæ and grains.

The patient was treated with "Disprin", and with the intramuscular injection of 800,000 units of "Cilicaine Forte" twice a day. On October 26, L.E. cells were found in the blood; the hæmoglobin value was 79%, the colour index was 1·0, the erythrocytes numbered 3,800,000 per cubic millimetre, the leucocytes numbered 3000 per cubic millimetre, and the erythrocyte sedimentation rate was 86 millimetres in the first hour. The joint pains disappeared and the rash faded a little after her admission to hospital, but a week later the joint pains had reappeared. The pyrexia failed to respond to penicillin, and "Achromycin" was substituted. Shortly after her admission, left lower lobe pneumonia developed; her temperature was swinging. Cortisone therapy was started on November 2. Soon afterwards, a left pleural effusion was noted. It had since been tapped on several occasions, but had reformed after each tapping. However, its size was less now than it had been originally. On November 29 she was put on prednisolone, ten milligrammes being given every six hours. On January 5, 1957, she developed acute cardiac failure and became anuric, but responded well to treatment. On January 7 she was put on a short trial of testosterone propionate, 25 milligrammes being given each day by intramuscular injection. Prednisolone was withdrawn. On January 23, with the return of the rash on her face, she was returned to prednisolone therapy. At the time of the meeting she was having five milligrammes three times a day.

Dr. Dodd said that in the past few weeks she had also developed an effusion on the right side of her chest, which had also been tapped on several occasions, and which had also reformed on each occasion. She had also had several attacks of pulmonary œdema. They had responded to treatment with aminophylline, given intravenously, and morphine, together with the intranasal administration of oxygen. She had had albuminuria for some weeks.

Chronic Alcoholism.

Dr. Dodd finally showed a male patient, aged sixty-two years, retired, who had been admitted to hospital on February 8, 1957, with a history of increasing shortness of breath of nine months' duration, and subsequently of œdema of the legs and abdominal distension. He had had no serious illness and no operation. He usually slept well, but complained of a troublesome cough with only a little sputum. More recently he had noticed œdema of the legs, abdominal distension, palpitations and anorexia, without nausea or vomiting. He had been a heavy drinker for thirty years, and his drinking had been even more excessive in the past few years.

On his admission to hospital, the patient's temperature was 98·8° F., his pulse rate was 108 per minute, and his respirations numbered 26 per minute. His blood pressure was 110 millimetres of mercury, systolic, and 75 millimetres, diastolic. Several spider nævi were present on the face and the upper part of the abdomen. The apex beat was in the fifth intercostal space 4·5 inches from the mid-sternal line. Faint dual rhythm was present, but there were no bruits. The lungs were clear anteriorly, but basal crepitations were heard posteriorly. The liver was enlarged three fingers' breadth below the costal margin. The abdomen was grossly distended and resonant to percussion into the flanks. There was an ulcer on the right leg, with cellulitis extending from the ankle to the knee. Gross pitting œdema was present in both legs. The reflexes were present in the upper limbs, but there were no ankle or knee jerks. The patient was confabulating and disorientated. Rectal examination revealed that the prostate was moderately enlarged, but there was no obliteration of sulci and the mucosa was mobile.

On February 9 the patient was incontinent of urine and fæces. Intermittent catheterization was instituted. An X-ray examination of the chest showed that the cardiac outline was much enlarged to right and left. The lungs appeared slightly congested. The hæmoglobin value was 86%. Liver function tests gave the following results: the thymol turbidity was one unit, the serum bilirubin content was five milligrammes per 100 millilitres, and the serum alkaline phosphatase content was 15 King-Armstrong units; the total protein content was 7·3 grammes *per centum* (albumin 4·2 grammes, globulin 3·1 grammes *per centum*), and the albumin-globulin ratio was 1·4:1·0. The response to the Wassermann test was negative.

The patient was treated by the administration of *Digitalis Folia*, one grain three times a day, "Neptal", two millilitres three times a week, "Diamox", one tablet on the days when "Neptal" was not given, and procaine penicillin, 600,000 units per day. A diet of high protein and low salt content was instituted, and fluids were restricted to two pints per day. Injections of "Combex" were given three times a week. Daily dressings were applied to the ulcer after cleansing with eusol. Good diuresis was obtained; the patient was able to pass urine unaided and with good control after intermittent catheterization for one week. The œdema had subsided, his mental condition had improved, and the ulcer was healing well. The abdominal distension had been relieved by the passage of a flatus tube and the administration of "Prostigmin". At the time of the meeting he was taking "Prostigmin" tablets twice a day.

Inadvertent Anaphylaxis.

DR. S. BRAND showed patients to illustrate various causes of anaphylactic reactions and their management and prevention. He said that anaphylaxis was one of the most frequently occurring therapeutic emergencies. Anaphylaxis might be (i) induced, from previous exposure to the allergen, or (ii) a natural or atopic sensitivity, when there was usually associated allergy often with strong hereditary influences; the patients were frequently horse-dander sensitive asthmatics. Anaphylaxis might occur with (*a*) administration of drugs and serum, (*b*) desensitization therapy, (*c*) skin-sensitivity testing procedures, (*d*) insect bites, (*e*) transfusions, (*f*) food intake in sensitive persons, and (*g*) investigations, such as excretion pyelography. The reaction might be immediate (anaphylactic shock) or delayed, occurring after six to twelve days (the serum-sickness type of reaction). The severe immediate type usually occurred within half an hour, and the earlier it occurred, the more likely was a fatal outcome. An onset with wheezing, sneezing and urticaria indicated a more favourable outcome.

Of the patients shown by Dr. Brand, the first had had severe anaphylactic shock within minutes of an intravenous injection of snake antivenene; no sensitivity tests had been performed. The second patient had had a similar attack after an intradermal test dose of undiluted horse serum prior to the administration of antivenene. All patients, on follow-up examination, were found to have skin sensitivity to diluted horse serum, the second patient reacting actively to a dilution of 1:1000. The third patient had had a severe reaction seven days after an injection of tetanus antiserum; skin testing had given negative results at the time of injury, but the result was now positive.

Dr. Brand went on to say that it seemed clear that sensitivity tests would definitely indicate those patients sensitive to horse serum who were likely to have severe and immediate anaphylactic reactions, though delayed reactions might not be detectable. Further tests should be carried out with diluted serum (at least 1:10) and by the scratch or intradermal method. In testing for allergens, scratch tests should usually be performed prior to intradermal testing. His fourth patient had had a moderately severe anaphylactic reaction a few minutes after an intradermal test with linseed extract. Careful checking of dosage, cautious increments and insistence that patients should wait at least twenty minutes after injections of potent extracts would prevent serious mishap during the treatment of allergic patients. The fifth patient had had an anaphylactic reaction with collapse three-quarters of an hour after an injection of pollen extract. Adrenaline was indicated in those cases, but often too much was given; 0·3 millilitre of 1:1000 solution under the site of the injection with a tourniquet above, and 0·3 to 0·6 millilitre of 1:1000 solution into the other arm were an adequate dose in most cases. Probably no more than 0·2 millilitre well diluted and given slowly should be administered intravenously. Large doses of antihistamines, given intramuscularly and intravenously, and in some cases steroids might be helpful. If shock was profound, supportive measures such as serum transfusion and the exhibition of oxygen and perhaps nor-adrenaline were indicated.

Cooley's Anæmia.

DR. H. D. STEWARD and DR. ALISON GARVEN showed a woman, aged twenty-eight years, who had been born in Cyprus, of Greek parentage. She presented in the eighth month of her fourth pregnancy with a hæmoglobin value of 6·5 grammes *per centum*, but with no symptoms of anæmia and no history of jaundice. At that time (October 1, 1954) a blood examination gave the following information: the red cells numbered 5,300,000 per cubic millimetre, the colour index was 0·4, the leucocytes numbered 10,000 per cubic millimetre, the hæmatocrit was 35%, the mean corpuscular volume was 66 cubic μ and the mean corpuscular hæmoglobin concentration was 19%. In a blood film the red cells showed gross anisocytosis, ranging from an occasional large polychromatic cell to numerous small microcytes. Many oval and target cells were present. There was hypochromia of all cells. The proportion of reticulocytes was 7%. The result of a Coombs test was negative. Fragility tests showed that hæmolysis began in 0·44% saline, but only 50% of hæmolysis had occurred at 0·28% (saline fragility was decreased). The serum iron content was 208 microgrammes per 100 millilitres. An X-ray examination of the skull, tibiæ and hands revealed no abnormality in the bony architecture. An investigation of the patient's family was carried out. The first child, aged seven years, had a hæmoglobin value of 10·8 grammes *per centum*; a blood film showed a few oval and target cells and a mild degree of hypochromia; the child was well. The second child, aged five years, had a hæmoglobin value of 11·2 grammes *per centum* and a normal blood film; the child was well. The third child, aged five and a half years, had a hæmoglobin value of 8·1 grammes *per centum*, and a blood film showed marked oval and target cell formation, anisocytosis and hypochromia; the child was well, but had had numerous respiratory tract infections. The patient's husband had a hæmoglobin value of 15 grammes *per centum*, and his blood film was normal.

During her pregnancy, the patient received four pints of blood. She was given two further transfusions during 1955.

Familial Splenomegaly.

Dr. Steward and Dr. Garven next showed a married woman, aged thirty-four years, who had attended a gynæcological clinic in August, 1953, when her spleen was found to be enlarged. She had no relevant symptoms. She had had acute nephritis in 1951, had suffered from varicose veins since the birth of her first child in 1947, and had had measles thirty-three years before. She had never been jaundiced, although other members of the family had been. An elder sister, aged forty-two years, had been found to have an enlarged spleen during an operation on an ovarian cyst. The eldest sister's son, aged eighteen years, also had a palpable spleen. A brother of the patient, aged thirty-six years, had a very large spleen, which frequently increased

in size and became tender during respiratory infections *et cetera*; he had had malaria. The patient's sons, aged eight and six years respectively, both had palpable spleens. The patient's daughter, aged nine months, had not a palpable spleen. A blood count on April 8, 1954, gave the following information: the hæmoglobin value was 12 grammes *per centum*, the erythrocytes numbered 3,760,000 per cubic millimetre, the leucocytes numbered 7500 per cubic millimetre, the hæmatocrit was 30%, the mean corpuscular volume was 79 cubic μ, and the mean corpuscular hæmoglobin concentration was 36%. Examination of a blood film revealed slight anisocytosis, with a few small cells, but the majority were normal in size and hæmoglobin content. Of the erythrocytes, 0·5% were reticulocytes. The result of a Coombs test was negative. Tests for fragility gave the following results: with normal blood, hæmolysis commenced at 0·42% saline and was complete at 0·32% saline; with the patient's blood, hæmolysis commenced at 0·40% saline and was complete at 0·30% saline.

Treatment with ferrous gluconate was begun, the dosage being five grains three times a day. On October 16, 1955, the patient's hæmoglobin value had fallen to 9·6 grammes *per centum*, and the proportion of reticulocytes was 2%; the patient was pregnant. After delivery the hæmoglobin value rose, and on January 31, 1957, it was 11·5 grammes *per centum*.

Myelomatosis.

Dr. Steward and Dr. Garven finally showed a male patient, aged sixty-nine years, who had been well until seven months prior to the meeting, when he collapsed at work; after that he noticed increasing weakness, dyspnœa and precordial pain on exertion, with swelling of the ankles. A blood count gave the following information: the hæmoglobin value was 5,200,000 grammes *per centum*, the erythrocytes numbered 2,000,000 per cubic millimetre and the leucocytes numbered 6000 per cubic millimetre. Nucleated red cells were seen in a blood film. Of the leucocytes, 60% were segmented neutrophile cells, 10% non-segmented neutrophile cells, 2% eosinophile cells, 16% lymphocytes and 12% monocytes. The blood findings suggested that the patient was suffering from leuco-erythroblastic anæmia. An X-ray examination of the bones revealed multiple small translucent areas, arousing suspicion, but not absolutely typical of myelomatosis. Bence-Jones proteose was present in the urine. A marrow biopsy revealed hypercellular marrow containing numerous myeloma cells. The total protein content of the serum was 6·7 grammes *per centum* (albumin 4·6, globulin 2·1, grammes *per centum*).

In view of the lack of symptoms, apart from those of anæmia, the patient was treated by blood transfusion only. After the initial transfusion his hæmoglobin level rose to 11·8 grammes *per centum*, but had fallen to 6·8% by February, 1957.

Ligation of the Cervix Uteri for Late Miscarriage.

DR. IAN A. MCDONALD gave a demonstration illustrating his method of ligating the *cervix uteri* to prevent miscarriage in cases of incompetence of the cervix. He showed that there were many causes of miscarriage which operated in the middle trimester of pregnancy, including fœtal abnormalities, placental insufficiency, hydramnios, fœtal death *in utero*, multiple pregnancy and congenital abnormalities of the uterus. Cervical incompetence was responsible for only a small number of repeated miscarriages in the middle trimester. Lacerations of the cervix during previous childbirth, surgical dilatation and amputation as part of a repair operation for prolapse were predisposing factors to the condition. In a few cases it appeared to be due to congenital laxity of the cervix.

Dr. McDonald said that cervical incompetence was suspected when the patient complained that she had miscarried on several occasions at an identical period of gestation. Usually the membranes had ruptured with little or no pain and the fœtus had been born alive. Suspected patients were examined vaginally with a speculum every week after pregnancy was first diagnosed. They were asked to report any vaginal discharge or abdominal discomfort or the presence of fullness in the vagina. When the cervix was seen to dilate and the membranes bulged through the internal os, the patient was taken to the operating theatre, and under a general anæsthetic, a purse-string suture of number 4 braided silk was placed around the exo-cervix at the level of the internal os. The herniating membranes were reduced and the suture was pulled tight enough to close the internal os completely. The patient rose next day and was discharged from the hospital the day following.

Dr. McDonald presented a series of charts illustrating the results obtained from 70 patients so treated. Thirty-three infants had been delivered from patients treated by cervical ligation. He said that when the method failed, it did so usually in the first few days after ligation. Even in successful cases the patients tended to come into premature labour when the stitch was divided. Otherwise it was removed at thirty-eight weeks of gestation. There usually followed a rapid and relatively painless labour.

Dr. McDonald emphasized the fact that careful selection of patients was imperative. Disaster might follow ligation of the cervix in a case in which incompetence was not the underlying cause.

Stress Incontinence of Urine.

DR. ROBERT ZACHARIN showed a schema for the interrogation of patients suffering from stress incontinence. He emphasized the importance of differentiating between patients with and without clinically evident prolapse. He discussed fully the management of patients without prolapse and the place of certain special tests in the investigation of such patients. The anatomical basis for a new therapeutic procedure, urethral suspension, was demonstrated on specimens.

Surgical Induction of Labour.

DR. G. A. MANLY presented, in the form of prepared slides, a summary of 500 surgical inductions of labour, covering the indications, the induction-delivery interval, the type of membrane rupture, the quality and quantity of liquor removed, the state of the cervix at the original rupture, the use of "Pitocin", the still-birth rate, the forceps rate and the Cæsarean section rate. Dr. Manly placed special emphasis on the following points. (i) Taken over all, 90% of women were delivered within seventy-two hours after artificial rupture of the membranes. (ii) There was no difference, whether the hind-waters or the fore-waters were ruptured. (iii) The quantity of liquor removed bore no relation to the induction-delivery interval. (iv) The quality of the liquor gave some indication of the outcome: (*a*) blood-stained liquor from hind-water puncture, necessitating a fore-water tap, usually indicated a rapid onset of labour; (*b*) milky liquor forecasted a rapid onset of labour and a baby of more than average size; (*c*) clear liquor, the usual finding, was of no value in predicting the outcome; (*d*) meconium staining at the original puncture did not call for immediate Cæsarean section. (v) The state of the cervix was important: (*a*) the more dilated it was, the sooner did labour ensue; (*b*) a closed cervix did not necessarily bode ill, as 77% of patients were delivered in three days, when the cervix was hard and unripe. (vi) The use of "Pitocin" after forty-eight hours from the rupture was successful in only 50% of cases. (vii) The still-birth, forceps and Cæsarean section rates were not increased after rupture of the membranes, although Cæsarean section was often performed as the easy way out in cases in which labour had not ensued after an arbitrary time.

Carcinoma of the Antrum Presenting as Chronic Sinusitis.

DR. J. R. THOMSON showed a woman, aged seventy-six years, who had presented with symptoms of unilateral (right) sinusitis, and who was found on examination to have a tumour in the right nasal cleft. X-ray examination of the sinuses revealed clouding of all the sinuses on the right side, and some destruction of the bony walls of the antrum. A biopsy revealed an adamantinoma.

Dr. Thomson stressed the need to exclude malignant disease in cases of apparent sinusitis in old people, particularly if the disease was unilateral. In relation to treatment, he said that it was necessary to take into consideration the patient's age, state of health and type, and the biological nature of the tumour and its extent. Treatment was by radical surgery (maxillary and ethmoid sinuses) through a Moure's lateral rhinotomy, followed by deep X-ray therapy.

Salivary Tissue Tumour.

Dr. Thomson next showed a female patient, aged forty-one years, who had a tumour of salivary tissue origin occupying and expanding the right tonsillar fossa. The histological picture resembled that seen in "cylindromata" of salivary glands. Treatment was by simple excision of the mass. The patient had been followed up for six months (up to the time of the meeting), and was so far free from recurrence.

X-Ray Films of Endoscopic Interest.

Dr. Thomson finally showed a number of X-ray films of endoscopic interest. They included films of chop bones, fish bones, coins and a drawing pin, all impacted in the upper part of the œsophagus. Other films were from the following patients: (i) a patient with massive collapse of the lung accompanied by hæmopneumothorax, who had presented at the ear, nose and throat department for bronchoscopy and aspiration of the bronchi; (ii) a patient with a lateral pharyngeal abscess following a rabbit-bone injury to the upper part of the œsophagus; the bone had apparently passed on after making a deep laceration in the right pyriform fossa area.

Transverse Lie of the Fœtus at Term and during Labour.

DR. J. M. BARRETT discussed transverse lie of the fœtus at term and during labour. He said that at the hospital, in the years 1955 and 1956, 2015 patients had been confined in the public wards. Among that number, there were six cases of transverse lie of the fœtus at term and during labour. Twin pregnancy was excluded from the series. The incidence ot transverse lie was one in 336 cases, a figure in keeping with reports from other hospitals. In those six cases, the ætiological factors were: (*a*) multiparity (two cases); (*b*) *placenta prævia* (three cases); (*c*) traumatic accidental hæmorrhage following attempted external cephalic version to correct a transverse lie (one case); that patient had an ovarian cyst occupying part of the pelvic cavity. Of the six patients, five had been delivered by Cæsarean section and one *per vaginam*. Four babies were alive and well, and two babies had died. Dr. Barrett thought that the proportion of fœtal deaths could be further reduced.

Dr. Barrett went on to say that ætiological factors in transverse lie of the fœtus were contracted pelvis (especially in *primigravidæ*), *placenta prævia*, multiparity, hydramnios, *fibromyomata uteri*, ovarian cysts, uterine malformations, and the cord tightly around the fœtus, especially the neck. The following were possible methods of management of such patients: (i) version: (*a*) external, (*b*) bipolar, (*c*) internal; (ii) Cæsarean section; (iii) decapitation (only when the patient had been neglected). It was considered that artificial rupture of the membranes after correction to a longitudinal lie was a dangerous procedure, because (*a*) there was a great danger of prolapse of the cord, (*b*) the malpresentation often recurred even after that procedure, and the situation had then greatly deteriorated through rupture of the membranes. Dr. Barrett suggested the following points in management: (i) Good ante-natal care and supervision of the patient. (ii) Adequate investigation of the possible ætiology, including soft-tissue placentography. (iii) Admission of the patient to hospital in the last weeks of pregnancy, and always to a hospital with adequate facilities. (iv) The performance of external cephalic version and then the application of pads and binder in cases in which contracted pelvis, *placenta prævia* and other obstructive lesions (fibromyomata and ovarian cysts) had been excluded. (v) If external version failed without anæsthesia, and if the patient was early in labour, attempted correction under anæsthesia, preferably in the operating theatre with preparations available for Cæsarean section. (vi) Limited use of bipolar and internal version. It should be remembered that 33% of cases of rupture of the uterus followed internal version. (vii) Widening of the indications for Cæsarean section.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE QUEENSLAND MEDICAL BOARD AND AMERICAN DIPLOMAS.[1]

[From *The Australasian Medical Gazette*, September, 1884.]

THE Queensland Medical Board at the instance of its President Dr. Bancroft has set a praiseworthy example of prudence and caution to the Medical Boards of the other colonies by passing a resolution refusing to register American diplomas unless the professional knowledge of their holders is certified to by the passage of an examination at either of the Universities of Melbourne or Sydney. It is much to be regretted that there is not sufficient backbone in the other colonies to take a similar firm stand. The longest time required by the American Colleges to be passed in study before the candidates are admitted for examination is three years, twelve months less than the shortest time required in the United Kingdom, whilst there are many colleges in the States who grant diplomas not only without study but without examination or even the presence of the candidate before the authorities of the Corporation granting the diploma. Neither do these Colleges require any proof of general education, so that it is possible for a man who cannot even read or write to become a fully fledged M.D. Most glaring examples of this easy road to the outward signs of knowledge in medicine are continually coming under our cognizance. Men without any previous training leave these Colonies for the United States and return in a few months with a diploma upon which they have the impudence to make application for registration as medical practitioners. In fact we know of one case in New South Wales in which a man who had never studied in and had never left that Colony obtained the degree of Doctor of Medicine from an American University and is now practising on the strength of it.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

SOME THOUGHTS ON THE CHEMOTHERAPY OF TUBERCULOSIS.

SIR: Dr. Bull is to be congratulated on producing an excellent summary of the present-day position of anti-tuberculosis chemotherapy (M. J. AUSTRALIA, May 25, 1957).

Whilst hesitating to join issue with one with such wide experience in this field, I feel that it may be of interest to mention the somewhat different viewpoint held at present by Professor J. W. Crofton and his colleagues in Edinburgh.

Failure to sterilize tuberculous lesions prior to surgery has been shown by Turnbull *et alii* (1955) to occur only when continuous chemotherapy along Medical Research Council recommended lines has been given for a period of less than eighteen months, when chemotherapy has been interrupted or inadequate, or when drug resistance was present. This is not to say that all cases should have chemotherapy of eighteen months' duration prior to resection.

Dr. Bull's group of therapeutic failures has another all too frequent cause. Group A (those excreting sensitive organisms) is seen frequently when patients fail to take the prescribed chemotherapy. This commonly occurs even in hospital in-patients being treated with PAS and INAH. Group B (those whose organisms become resistant) are almost invariably due to taking chemotherapy irregularly, such as receiving streptomycin and taking PAS or INAH on rare occasions. In this regard the work of J. McD. Simpson is relevant. He found that when the urine of out-patients believed to be taking PAS was tested for this drug without prior warning, in more than 30% of cases a negative result was obtained. The corresponding figure for in-patients was not much lower. If Medical Research Council recommended chemotherapy is prescribed for a patient with an infection due to sensitive organisms, and is regularly taken, the development of drug resistance, and indeed failure of sputum conversion, is exceptional.

Caution is advisable in prescribing buffered PAS and PAS resin complexes. It must be remembered that these compounds have not yet been shown in controlled trial in combination with streptomycin or INAH to be effective in preventing the development of drug resistance. It is possible that they are better tolerated by virtue of a lower blood level due to incomplete absorption.

"Terramycin" can safely be given to the majority of patients for long periods in the requisite dosage of three to five grammes daily. Murdoch and Stewart (1956) found that in only one of 21 patients was a three months' course not tolerated. There are a number of patients in Edinburgh who have taken four or five grammes of this drug daily for twelve months without any notable side effects developing. It is true, however, that "Terramycin" is seldom effective in delaying the emergence of drug-resistant organisms for

a long period when given in conjunction with streptomycin or INAH.

Presumably Dr. Bull was not advocating the administration of INAH alone in "benign" forms of tuberculosis. The very term "benign" implies the presence of a low bacterial population; but this is not to say that sufficient organisms may not survive therapy to initiate a less benign form of the disease, which may then fail to respond to INAH.

Yours, etc.,

2 Collins Street, Melbourne,
May 30, 1957.

BRIAN MARKS.

References.

MURDOCH, J. McC., and STEWART, S. M. (1956), "Viomycin with Oxytetracycline in the Treatment of Pulmonary Tuberculosis", *Brit. J. Tuberc.*, 50: 85.

SIMPSON, J. McD. (1956), personal communication.

TURNBULL, F. W. A., STEWART, S. M., and MACGREGOR, A. R. (1955), unpublished observations.

TRAINING THE MEDICAL STUDENT FOR FAMILY COUNSELLING.

SIR: I am extremely interested in the article by Dr. Leigh Cook in your issue of May 25, "Training the Medical Student for Family Counselling". May I say that in my experience, the doctor who seeks to be understanding and sympathetic has perforce to deal with problems which, on close questioning, will be found to properly belong to the province of a minister of religion. It is quite true that patients will consult their doctor rather than their minister; the hypertension of a patient of mine I hope to relieve by calling the minister to assist me to treat her drunkard son, thus relieving her worries.

Perhaps I may be forgiven for being biased, but I believe that "Physician, heal thyself" does apply to us; for how can one give help to a patient with a primarily spiritual problem, unless one knows, as an experimental fact, the personal presence of One Who is "a very present help in trouble"? I believe that the education of students, and indeed our own education, is incomplete unless we take care to include the spiritual side.

Yours, etc.,

41 Marlborough Street, Brighton,
South Australia.
May 29, 1957.

JOHN B. HUGHES.

THE EARLY TREATMENT OF SQUINT.

SIR: Every ophthalmic surgeon will agree with Dr. B. A. E Harley ("The Early Treatment of Squint", M. J. AUSTRALIA, May 11, 1957) that the early treatment of squint in childhood is imperative. Only in this way can suppression amblyopia in the deviating eye be prevented or relieved. Like most other writers on this subject, Dr. Harley passes over the occurrence of congenital amblyopia, a condition commonly found in one eye. Its possessors behave in much the same fashion as the hypermetropes mentioned by Dr. Harley—some develop a squint, others remain straight and are usually quite unaware of their condition until it is picked up by the school medical officer. A similar condition is often found to be present in other members of the family with or without a squint. In these cases occlusion of the good eye is useless, and failure to appreciate the exact nature of the amblyopia results in subjecting the unfortunate child to months of torture by occluding the good eye.

As many individuals with one amblyopic eye never develop strabismus, it can hardly be claimed that the possession of full binocular vision is the factor that keeps eyes straight. When the vision of one eye is lost in adult life, the future behaviour of the blind eye is unpredictable—some deviate, others remain straight. Conversely, not all squinters develop amblyopia in the deviating eye.

There is a large, but unfortunately inarticulate, body of opinion amongst ophthalmic surgeons that orthoptic treatment is useless. T. Keith Lyle, an English ophthalmic surgeon well known for his voluminous writings on squint for the past twenty years, has stated recently (Lyle and Foley, 1957) that "Great care must be taken in the selection of patients suitable for orthoptic treatment, particularly in the case of non-paralytic non-accommodative convergent strabismus". As he also indicates that "full binocular single vision" is attained in accommodative strabismus without the use of orthoptics, it is difficult to understand why he advocates this form of treatment at all. The pursuance of a course of orthoptic training may mean a series of regular visits to the orthoptist over a period of many months. Apart from the expense, there is the great inconvenience and loss of time to the busy and anxious mother of a family willing to do anything to relieve her child's deformity.

When the time is ripe for operation, it is unreasonable to suppose that any degree of accuracy can be expected by operating on a muscle which is in a state of spasm and which relaxes under the anæsthetic. The wonder is that so often the squint is relieved by operation. What is likely to happen one, or ten, or twenty years later is another story. In these days of shortage of hospital beds, we have come to realize that prolonged stay in hospital is unnecessary. Sufficient time should be allowed for the child to recover from the anæsthetic, and only the operated eye need be covered. Even that dressing may be removed after twenty-four hours. Bandaging of both eyes for a week and the daily attendance of the surgeon armed cap-à-pie in sterile gown, cap, mask and gloves is surely an anachronism.

Finally, it is not difficult to agree with Dr. Harley that the criterion of cure in the squint of childhood is the attainment of full binocular vision. Every surgeon of experience will agree with Lyle that too often that ideal is not attained, and that the result is cosmetic only. Nevertheless, all is not lost. The little squinter may grow up to make a big score at a test match or to land an aircraft safely.

Yours, etc.,

33 Collins Street, Melbourne,
May 24, 1957.

KEVIN O'DAY.

Reference.

LYLE, T. K., and FOLEY, J. (1957), "Prognosis in Cases of Strabismus with Special Reference to Orthoptic Treatment" *Brit. J. Ophth.*, 41: 129.

Obituary.

ELEANOR ELIZABETH BOURNE.

DR. ELEANOR ELIZABETH BOURNE, who had not long ago completed fifty years' membership of the Queensland Branch of the British Medical Association, and had been elected an honorary life member of the Branch, died in Brisbane on May 23, 1957. She was aged seventy-nine years. Dr. Bourne was born in Brisbane and was educated at the Brisbane Girls' Grammar School and the University of Sydney, to which she received an exhibition in 1895, being the first woman student to receive such an exhibition from the Queensland Government. In 1903 she graduated in medicine and surgery, and returned to Brisbane to become the first woman junior resident medical officer at the Brisbane General Hospital. She later became Superintendent of the Brisbane Children's Hospital, and from 1910 to 1914 held the position of Medical Inspector of State Schools in Queensland. In 1920 she became Assistant Medical Officer to the city of Carlisle in England, and did not return to Brisbane until 1938. Dr. Bourne was founder-president of the Standing Committee of the Women's College of the University of Queensland and had recently made a generous gift to the College building fund. She had been living in retirement for some time prior to her death.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Examination Results.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the undermentioned candidates satisfied the examiners at the recent examinations for Part II of the various medical diplomas of the University of Sydney as shown below.

Anæsthesia: Loraine C. Hibbard, Helen M. Windon.

Dermatological Medicine: E. Kocsard, M. O'Loughlin, K. G. Poyzer.

Laryngology and Oto-Rhinology: O. J. Davies.

Ophthalmology: H. A. Handley, Phyllis M. Waddy.

Psychological Medicine: Lesley H. Barnes, J. H. T. Ellard, J. Grady.

Diagnostic Radiology: J. D. Cashman.

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

PROGRAMME FOR JULY, 1957.

Week-End Course at Camperdown.

A WEEK-END COURSE will be held at Camperdown on July 20, 1957. The programme is as follows: 3.45 p.m., "Bronchial Asthma", Dr. V. G. Bristow; 5.30 p.m., "Diabetes and Hypertension in Pregnancy", Dr. W. McI. Rose. The local secretary for this course is Dr. R. R. Sobey, 102-104 Liebig Street, Warrnambool (telephone 317).

Course in Pathology for the M.C.R.A. and D.D.R.

On Tuesdays, at 4.30 p.m., there will be the following series of lectures in pathology suitable for candidates for M.C.R.A. and D.D.R.: July 2, "Tumours and Malformations of the Urinary Tract", Dr. J. S. Peters, at Prince Henry's Hospital Lecture Theatre; July 9, "Pathology of the Jaws", Dr. Julian Reich, at the Dental Hospital; July 16, "Tumours of the Alimentary Tract", Dr. Rowan Webb, at the Royal Melbourne Hospital, Radiology Department; July 23, "Pathology of the Lungs and Pleura", Dr. Malcolm Allen, at the Alfred Hospital, Auxiliary Room; July 30 and August 6, "Tumours of Bone", Dr. R. Kaye Scott, in the lecture room, 278 William Street, C.1.

The fee for this course is £5 5s. Those already enrolled for basic pathology need pay no further fee.

Visit of Professor R. J. Kellar.

Full details will be published shortly regarding the visit to Melbourne from July 27 to August 10 of Professor R. J. Kellar, Professor of Obstetrics and Gynæcology, University of Edinburgh, the Category A. Lecturer for 1957 of the Australian Post-Graduate Federation in Medicine. Professor Kellar will lecture for the committee at 8.15 p.m. on July 30 and on August 1, 6 and 8, and to the Royal College of Obstetricians and Gynæcologists on July 31. He will also make a number of visits to hospitals.

INFORMATION.

The address of the Melbourne Medical Post-Graduate Committee is 394 Albert Street, East Melbourne, C.2. Telephone: FB 2547.

The College of General Practitioners.

VICTORIA FACULTY.

THE first annual meeting of the Victoria Faculty of The College of General Practitioners is to be held at the rooms of the Victorian Branch of the British Medical Association, Albert Street, East Melbourne, on July 11, 1957, at 8 p.m. The business of the meeting is to confirm the by-laws of the Faculty, elect the Board of the Faculty for the ensuing year, and receive the annual report, which includes reports from the subcommittees for undergraduate and post-graduate education and research in general practice. After this business has been completed, the meeting will be addressed by Professor R. Lovell, Professor of Medicine in the University of Melbourne, an Associate member of the Faculty.

All members of the British Medical Association are welcome to attend this meeting, when the activities of the College will be described and opportunities will be available to apply for membership and associateship.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MAY 25, 1957.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	4(1)	6(5)	8(2)	..	1(1)	..	..	..	19
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	12(11)	12(12)	..	..	..	..	..	..	24
Diphtheria	..	5(5)	..	..	4(3)	2	..	..	11
Dysentery (Bacillary)	..	..	3(1)	..	..	..	..	..	3
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	40(18)	24(6)	..	2(2)	4(2)	..	2	1	73
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	3	..	..	..	4	..	3 4
Malaria	..	..	..	..	..	..	..	..	1
Meningococcal Infection	..	..	1(1)	..	1	..	..	..	1
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	2	..	1	1	..	1	..	..	5
Puerperal Fever	..	..	..	1(1)	..	..	..	..	1
Rubella	..	15(12)	..	10(3)	9(3)	..	..	..	34
Salmonella Infection	..	..	..	2(2)	..	..	..	..	2
Scarlet Fever	12(8)	22(16)	5(3)	3(3)	2(1)	..	..	..	44
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	2(1)	..	..	..	2
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	22(14)	14(8)	18(5)	3(1)	11(7)	3	9	..	80
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes *et cetera* are promulgated in the *Commonwealth of Australia Gazette*, Number 25, of May 9, 1957.

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—1/39200 Captain N. C. Davis is appointed from the Reserve of Officers, 22nd February, 1957. The following officers are appointed from the Reserve of Officers and to be Captains (provisionally), 22nd February, 1957: Honorary Captains 1/39204 H. R. Withers and 1/39205 J. P. Waller. 1/66025 Captain (provisionally) R. M. Goodwin relinquishes the provisional rank of Captain, 18th February, 1957, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps) (Medical) (Northern Command), and is granted the honorary rank of Captain, 19th February, 1957. 1/43713 Lieutenant-Colonel A. Fryberg, M.B.E., is placed upon the Retired List (Northern Command) with permission to retain his rank and wear the prescribed uniform, 27th May, 1957. To be Lieutenant-Colonel, 1st October, 1956: 1/39147 Major (Temporary Lieutenant-Colonel) C. C. Wark, M.B.E. To be Major, 7th March, 1957: 1/39130 Captain P. J. F. Grant.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—The provisional appointment of 2/108252 Captain D. P. Clarke is terminated, 13th March, 1957. 2/147971 Captain L. E. Jabour is appointed from the Reserve of Officers, 7th March, 1957. 2/167654 Captain L. L. Nordstrom is transferred to the Reserve of Officers (Royal Australian Army Medical Corps) (Medical) (Eastern Command), 10th January, 1957. 2/143608 Captain (provisionally) C. S. H. Reed relinquishes the provisional rank of Captain, 27th February, 1957, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps) (Medical) (Eastern Command) and is granted the honorary rank of Captain, 28th February, 1957. To be Captains (provisionally): 2/79238 Richard Michael McCredie, 6th February, 1957, 2/79249 Ronald Charles Wonson, 28th February, 1957, and 2/108252 Desmond Patrick Clarke, 14th March, 1957.

Western Command.

Royal Australian Army Medical Corps (Medical).—5/26514 Captain R. Paton ceases to be seconded whilst in the United Kingdom, 1st February, 1957. To be Captains (provisionally): 5/26559 Peter Maxwell Connor and 5/26560 Donald Golinger, 14th March, 1957, and 5/38067 Byron Arthur Kakulas, 15th March, 1957.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

Northern Command.—To be Honorary Captains: Geoffrey Donald Broadbent, 22nd February, 1957, Thomas Blair Buchanan, 25th February, 1957, and George Chong Wah and Mervyn Cheong, 15th March, 1957.

Southern Command.—Honorary Captains C. V. Childs and P. Goodman are retired, 12th March, 1957. The resignation of Captain K. E. Harrison of his commission is accepted, 10th December, 1956. To be Honorary Captain, 4th February, 1957: Janus Mederic Gaetan Fraillon.

The following officer is placed upon the Retired List (Southern Command) with permission to retain his rank and wear the prescribed uniform, 12th March, 1957:

Southern Command: Captain A. E. Lincoln.

HONOURS AND AWARDS.

THE following award was made in connexion with the Australian Military Forces:

The Australian Efficiency Decoration: 5/26391 Colonel Rowland Ralph Anderson, M.C., Royal Australian Army Medical Corps (Medical).

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

McJannet, Alan Hugh Dalton, M.B., B.S., 1947 (Univ. Sydney), 217 Macquarie Street, Sydney.

Deaths.

THE following deaths have been announced:

HIRSCHFELD.—Otto Saddler Hirschfeld, on May 29, 1957, at Brisbane.

HART.—Basil Lloyd Hart, on May 30, 1957, at Southport, Queensland.

MCCLELLAND.—William Caldwell McClelland, on May 30, 1957, at Middle Brighton, Victoria.

CAWTHORN.—Frank Raymond Cawthorn, on May 31, 1957, at Mooroopna, Victoria.

FREEMAN.—William John Freeman, on June 3, 1957, at Sydney.

Diary for the Month.

JUNE 17.—Victorian Branch, B.M.A.: Finance Subcommittee.
JUNE 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 19.—Western Australian Branch, B.M.A.: General Meeting.
JUNE 20.—South Australian Branch, B.M.A.: Scientific Meeting.
JUNE 20.—Victorian Branch, B.M.A.: Executive of Branch Council.
JUNE 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 25.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 88 L'Estrange Terrace, Kelvin Grove, Brisbane): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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